```
FILE 'HCAPLUS' ENTERED AT 13:30:24 ON 22 JUL 2008
           3287 S URSODEOXYCHOL?
1.2
         139310 S ISCHEM? OR STROKE OR NEUROPROTECTIVE
1.3
             28 S L1 AND L2
L4
             19 S L3 AND (PY<2005 OR AY<2005 OR PRY<2005)
     FILE 'HCAPLUS' ENTERED AT 15:30:51 ON 22 JUL 2008
         96359 S PREBIOTIC OR ENTERAL OR DIARRHEA OR NUTRITIONAL
L6
         493913 S ADHESIVE OR ADHESION
    FILE 'HCAPLUS' ENTERED AT 15:31:22 ON 22 JUL 2008
L7
         95742 S ((ARABINO OR MANNO OR GALACTO OR ISOMALTO OR SIALYL) (W) OLIGOS
L8
             19 S L5 AND L6 AND L7
    FILE 'HCAPLUS' ENTERED AT 15:32:11 ON 22 JUL 2008
1.9
              8 S L8 AND (PY<2004 OR AY<2004 OR PRY<2004)
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L10
           2473 S HT29
L11
              1 S L5 AND L6 AND L10
L12
              1 S L5 AND L7 AND L10
L13
              0 S L11 AND (PY<2004 OR AY<2004 OR PRY<2004)
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              0 S L12 AND (PY<2004 OR AY<2004 OR PRY<2004)
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L15
             26 S L5 AND L10
           188 S L6 AND L10
L16
            15 S L15 AND (PY<2004 OR AY<2004 OR PRY<2004)
L17
L18
           115 S L16 AND (PY<2004 OR AY<2004 OR PRY<2004)
    FILE 'REGISTRY' ENTERED AT 16:40:01 ON 22 JUL 2008
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L19
              1 S E4
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                EXP ALPHA 1,3-MANNOBIOSE/CN
                EXP ALPHA 1.3 MANNOBIOSE/CN
                EXP 1,3 MANNOBIOSE/CN
                EXP ALPHA 1,2-MANNOBIOSE/CN
                EXP ALPHA 1,6-MANNOBIOSE/CN
                EXP MANNOOLIGOSACCH/CN
    FILE 'CAPLUS' ENTERED AT 16:41:54 ON 22 JUL 2008
L20
             0 S L19/THU
L21
             1 S L19
L22
           380 S MANNOBIOSE
    FILE 'REGISTRY' ENTERED AT 16:43:59 ON 22 JUL 2008
L23
             1 S MANNOBIOSE/CN
     FILE 'CAPLUS' ENTERED AT 16:44:08 ON 22 JUL 2008
L24
              6 S L23/THU
    FILE 'HCAPLUS' ENTERED AT 16:45:21 ON 22 JUL 2008
L25
             64 S METHYL ALPHA MANNO?
L26
          74681 S NUTRITIONAL OR ENTERAL OR PREBIOTIC
L27
              0 S L25 AND L26
     FILE 'STNGUIDE' ENTERED AT 16:46:00 ON 22 JUL 2008
    FILE 'HCAPLUS' ENTERED AT 16:46:41 ON 22 JUL 2008
T.28
        714245 S GUT OR INTESTINE OR ORAL OR PHARMACEUTICAL
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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4 FILE LAST UPDATED: 20 Jul 2008 (20080720/ED)

HCAplus now includes complete International Patent Classification (IPC) reclassification data for the second quarter of 2008.

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This file contains CAS Registry Numbers for easy and accurate substance identification.

>> s ((arabino or manno or galacto or isomalto or sialyl)(w)oligosaccharide)) or arabinooligosaccharide or mannooligosaccharide or galactooligosaccharide or isomaltooligosaccharide or sialyloligosaccharide or pectin or lactose or curdlan or lactulose or (beta(2a)glucan)

UNMATCHED RIGHT PARENTHESIS 'ACCHARIDE') OR'
The number of right parentheses in a query must be equal to the number of left parentheses.

=> s prebiotic or enteral or diarrhea or nutritional

4374 PREBIOTIC 4337 ENTERAL

22140 DIARRHEA

66844 NUTRITIONAL

96359 PREBIOTIC OR ENTERAL OR DIARRHEA OR NUTRITIONAL

=> s adhesive or adhesion

224861 ADHESIVE 325612 ADHESION

L6 493913 ADHESIVE OR ADHESION

=> file stnguide

L5

COST IN U.S. DOLLARS SINCE FILE TOTAL SESSION ENTRY FULL ESTIMATED COST 2.69 64.26 DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) SINCE FILE TOTAL. ENTRY SESSION CA SUBSCRIBER PRICE 0.00 -15.20 FILE 'STNGUIDE' ENTERED AT 15:30:54 ON 22 JUL 2008 USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT COPYRIGHT (C) 2008 AMERICAN CHEMICAL SOCIETY (ACS)

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jul 18, 2008 (20080718/UP).

=> file hcaplus

 COST IN U.S. DOLLARS
 SINCE FILE TOTAL ENTRY
 TOTAL SESSION

 FULL ESTIMATED COST
 0.06
 64.32

FILE 'HCAPLUS' ENTERED AT 15:31:22 ON 22 JUL 2008
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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4 FILE LAST UPDATED: 20 Jul 2008 (20080720/ED)

 ${\tt HCAplus}$ now includes complete International Patent Classification (IPC) reclassification data for the second quarter of 2008.

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

»> s ((arabino or manno or galacto or isomalto or sialyl) (w)oliqosaccharide) or arabinooliqosaccharide or mannooliqosaccharide or galactooliqosaccharide or isomaltooliqosaccharide or or isomaltooliqosaccharide or curdlan or lactulose or (beta(2a)clucan)

- 4078 ARABINO
- 2731 MANNO
- 3051 GALACTO
- 213 ISOMALTO
- 4660 SIALYL
- 32176 OLIGOSACCHARIDE 289 (ARABINO OR MANNO OR GALACTO OR ISOMALTO OR SIALYL)(W)OLIGOSACCH
 - 10 ARABINOOLIGOSACCHARIDE
 - 213 MANNOOLIGOSACCHARIDE
 - 417 GALACTOOLIGOSACCHARIDE
 - 344 ISOMALTOOLIGOSACCHARIDE
 - 154 SIALYLOLIGOSACCHARIDE

26974 PECTIN 58407 LACTOSE

1309 CURDLAN 2245 LACTULOSE

1544839 BETA

16163 GLUCAN 8179 BETA(2A)GLUCAN

L7 95742 ((ARABINO OR MANNO OR GALACTO OR ISOMALTO OR SIALYL) (M)OLIGOSACC
HARIDE) OR ARABINOOLIGOSACCHARIDE OR MANNOOLIGOSACCHARIDE OR
GALACTOOLIGOSACCHARIDE OR ISOMALTOOLIGOSACCHARIDE OR SIALYLOLIGO
SACCHARIDE OR PECTIN OR LACTOSE OR CURDLAN OR LACTULOSE OR (BETA
(2A) GLUCAN)

=> s 15 and 16 and 17

L8 19 L5 AND L6 AND L7

=> file stnguide

COST IN U.S. DOLLARS SINCE FILE TOTAL SESSION ENTRY FULL ESTIMATED COST 2.69 67.01 TOTAL DISCOUNT AMOUNTS (FOR OUALIFYING ACCOUNTS) SINCE FILE ENTRY SESSION CA SUBSCRIBER PRICE 0.00 -15.20

FILE 'STNGUIDE' ENTERED AT 15:31:26 ON 22 JUL 2008 USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT COPYRIGHT (C) 2008 AMERICAN CHEMICAL SOCIETY (ACS)

FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: Jul 18, 2008 (20080718/UP).

=> file hcaplus

 COST IN U.S. DOLLARS
 SINCE FILE ENTRY
 TOTAL ENTRY

 FULL ESTIMATED COST
 0.06
 67.07

 DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
 SINCE FILE TOTAL ENTRY SESSION 0.00
 TOTAL ENTRY SESSION 0.00

FILE 'HCAPLUS' ENTERED AT 15:32:11 ON 22 JUL 2008
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4 FILE LAST UPDATED: 20 Jul 2008 (20080720/ED) HCAplus now includes complete International Patent Classification (IPC) reclassification data for the second quarter of 2008.

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s 18 and (PY<2004 or AY<2004 or PRY<2004)

23986246 PY<2004 4779965 AY<2004

4250851 PRY<2004

L9 8 L8 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnguide

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FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: Jul 18, 2008 (20080718/UP).

=> d 19 1-8 ti abs bib
YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:v

- L9 ANSWER 1 OF 8 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Oligosaccharide-containing nutritional compositions that inhibit

pathogen adhesion to intestinal cells

- AB Saccharides (particularly oligosaccharides) are used as inhibitors of pathogen adhesion to mammalian cells (especially gut cells) and may be used in food and nutritional compns. Compds. are screened for inhibition of adhesion of specific pathogens (verocytotoxic and enteropathogenic Escherichia coli) to the colonic epithelium (HT 29 cell line) without adversely affecting the colonic microflora or adhesion of probiotic organisms. Compds. with suitable activity include mannooligosaccharides, caseinoglycomacropeptides, chitooligosaccharides, glastcooligosaccharides, etc.
 - 2005:283268 HCAPLUS <<LOGINID::20080722>>
- AN 2005:28326 DN 142:335365
 - II Oligosaccharide-containing nutritional compositions that inhibit pathogen adhesion to intestinal cells
- IN Rhoades, Jonathan Robert; Rastall, Robert; Gibson, Glenn R.
- PA Novartis Ag, Switz.
- SO PCT Int. Appl., 31 pp. CODEN: PIXXD2
- DT Patent
- LA English

FAN CNT

AB

FAN.	PAT	1 ENT				KIN	D	DATE			APPL	ICAT	ION	NO.		D	ATE		
PI	WO	2005	0276	63		A2 A3		2005			WO 2	004-	EP10	469				917 <	
		W:	AE, CN, GE, LK, NO, TJ, BW, AZ, EE, SI,	AG, CO, GH, LR, NZ, TM, GH, BY, ES,	AL, CR, GM, LS, OM, TN, GM, KG, FI,	AM, CU, HR, LT, PG, TR, KE, KZ,	AT, CZ, HU, LU, PH, TT, LS, MD, GB,	AU, DE, ID, LV, PL, TZ, MW, RU, GR, CF,	DK, IL, MA, PT, UA, MZ, TJ, HU,	DM, IN, MD, RO, UG, NA, TM, IE,	DZ, IS, MG, RU, US, SD, AT, IT,	EC, JP, MK, SC, UZ, SL, BE, LU,	EE, KE, MN, SD, VC, SZ, BG, MC,	EG, KG, MW, SE, VN, TZ, CH, NL,	ES, KP, MX, SG, YU, UG, CY, PL,	FI, KR, MZ, SK, ZA, ZM, CZ, PT,	GB, KZ, NA, SL, ZM, ZW, DE, RO,	GD, LC, NI, SY, ZW AM, DK, SE,	
PRAI	US GB	2004 2006 2003 2004	0039 0287 -219	79 276 96		A		2006 2006 2003 2004	1221 0919		BR 2 US 2							920 < 320 <	

L9 ANSWER 2 OF 8 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Prebiotic oligosaccharides: evaluation of biological activities

and potential future developments A review. Prebiotics are recognized for their ability to increase levels of 'health promoting' bacteria in the intestinal tract of humans or animals. This normally involves targeting the activities of bifidobacteria and/or lactobacilli. Non digestible oligosaccharides such as fructo-oligosaccharides, lactulose and traps-galactooligosaccharides seem to be efficacious prebiotics in that they confer the degree of selective fermentation required. Other oligomers are used as prebiotics in Japan e.g. xylo-oligosaccharides, soybean-oligosaccharides, isomalto-oligosaccharides. To determine prebiotic functionality, various in vitro systems may be used. These range from simple batch culture fermenters to complex models of the gastrointestinal tract. The definitive test however is an in vivo study. The advent of mol. based procedures in gut microbiol. has alleviated many concerns over the reliability of microbial characterization, in response to prebiotic intake. Techniques such as DNA probing and mol. fingerprinting are now being applied to both laboratory and human studies. These will help to further identify prebiotics that can be added to the diet and thereby fortify 'beneficial' bacteria. Such robust technologies can also be used in structure-function assays to identify the mechanisms behind prebiotic effects. Considerable research effort is currently being expended in developing so called 'second generation' prebiotics. These are forms that have multiple biol. activity that attempts health enhancement properties beyond the genus level stimulation of bifidobacteria or lactobacilli within the gut microbflora. Examples include higher mol. weight oligomers than is conventional for prebiotics, such that targeted activities in the distal colon are feasible (the left side of the human large gut being the frequent area for colonic disorder). Glycobiol. is also developing anti-adhesive prebiotics that incorporate receptor sites for common gut pathogens and/or their activities. Through the use of reverse enzyme technol., as applied to β-galactosidase activity in prebiotics, oligosaccharides that enhance a lactic microflora at the species, rather than genus, level are possible. This review gives an account of how second generation prebiotics may be manufactured, through a variety of biotechnol. techniques, and tested for their biol. activity. The health attributes of such mols. as well as existing prebiotics is also discussed, with reference to specific target populations. The prebiotic concept is a much more recent development in

dietary intervention for enhanced gut function than is prebiotics. Not surprisingly therefore, research developments are proceeding quickly. Because oligosaccharides can be added to a wide variety of foodstuffs, new functional food developments are continuing. It is important that these are tested using reliable methodologies and that any health effects are underpinned by realistic mechanisms of effect.

- AN 2002:783388 HCAPLUS <<LOGINID::20080722>>
- DN 138:168911
- TI Prebiotic oligosaccharides: evaluation of biological activities and potential future developments
- AU Rastall, Robert A.; Gibson, Glenn R.
- CS Unit of Food Microbial Sciences, School of Food Biosciences, University of Reading, Reading, RG6 6AP, UK
- SO Probiotics and Prebiotics (2002), 107-148. Editor(s): Tannock, Gerald W. Publisher: Caister Academic Press, Wymondham, UK. CODEN: 69DEL7; ISBN: 0-9542464-1-1
- DT Conference; General Review
- LA English
- RE.CNT 99 THERE ARE 99 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L9 ANSWER 3 OF 8 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI The sialylated fraction of milk oligosaccharides is partially responsible for binding to enterotoxigenic and uropathogenic Escherichia coli human strains
- AB Milk oligosaccharides can act as soluble receptors that block bacterial adhesion to the different epithelia. Colonization factor antigens (CFA)/I- and CFA/II-expressing enterotoxigenic Escherichia coli (ETEC) strains constitute one of the main causes of diarrhea in infants. Here, the inhibition of hemagglutination mediated by these strains by milk oligosaccharides was tested. Human milk oligosaccharides showed a strong inhibitory capacity, which decreased when the oligosaccharides were desialylated. Because milk oligosaccharides also are present in the urine of neonates receiving mothers' milk, their ability to bind two uropathogenic Escherichia coli (UPEC) strains was also examined UPEC strains expressing P (Pap) and P-like (Prs) fimbriae are responsible for infections of the urinary tract such as pyelonephritis and cystitis. The hemagglutination mediated by these strains was inhibited by human milk oligosaccharides. The sialylated fraction was partially responsible for this inhibition in the case of the UPEC expressing the P-like fimbria because differences were found after desialvlation. Although bovine milk oligosaccharides were less efficient at inhibiting the hemagglutination of ETEC strains, they were still quite good inhibitors of UPEC strains.
- AN 2002:779738 HCAPLUS <<LOGINID::20080722>>
- DN 138:24118
- TI The sialylated fraction of milk oligosaccharides is partially responsible for binding to enterotoxigenic and uropathogenic Escherichia coli human strains
- AU Martin-Sosa, Samuel; Martin, Maria-Jesus; Hueso, Pablo
- CS Departamento de Bioquimica y Biologia Molecular, Facultad de Biologia, Universidad de Salamanca, Salamanca, 37007, Spain
- SO Journal of Nutrition (2002), 132(10), 3067-3072
- CODEN: JONUAI; ISSN: 0022-3166
- PB American Society for Nutritional Sciences
- DT Journal
- LA English
- RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L9 ANSWER 4 OF 8 HCAPLUS COPYRIGHT 2008 ACS on STN

- Blocking adhesion of pathogenic microorganisms to avian cells For the production of a pharmaceutical preparation for the blocking of adhesion of germs to bird cells one may use oligogalacturonides with a polymerization degree ≥ 2 and a degree of esterification < 20% as active substance, optionally together with an ordinary pharmaceutical carrier, auxiliary substances, and fillers into a form suitable for
 - administration to poultry. 2002:446014 HCAPLUS <<LOGINID::20080722>>
- DN 137:15762

AN

SO

- TI Blocking adhesion of pathogenic microorganisms to avian cells
- IN Guggenbichler, Josef Peter; Jurenitsch, Johann
- de Bettignies-Dutz, Andreas, Germany PA
- Ger. Offen., 12 pp. CODEN: GWXXBX
- DT Patent

LA	Ger	cman	
FAN.	CNT	1	
	י מכו	TIME	

	PATENT NO.									APPLICATION NO.								
PI		1006157 2002047					2002 2002										211 < 211 <	
		GM LS PL	, CR, , HR, , LT,	CU, HU, LU, RO,	CZ, ID, LV, RU,	DE, IL, MA, SD,	DK, IN, MD, SE,	DM, IS, MG, SG,	DZ, JP, MK, SI,	EC, KE, MN, SK,	EE, KG, MW,	ES, KP, MX,	FI, KR, MZ,	GB, KZ, NO,	GD, LC, NZ,	GE, LK, OM,	GH, LR, PH,	
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		R: AT IE	, BE,									LI,	LU,	NL,	SE,	MC,	PT,	
PRAI	RU DE	326974 2281104 2000-10 2001-EP	06157	4	C2 A		2006 2000	0810 1211	<-	RU 2 -							211 < 211 <	
	wO	Z001-EP	T404T		W		7001	1711	<-	_								

- 1.9 ANSWER 5 OF 8 HCAPLUS COPYRIGHT 2008 ACS on STN
- ΤI Methods for drug administration and distribution based on monitoring blood viscosity and other parameters for diagnostics and treatment
- AB Various methods are provided for determining and utilizing the viscosity of the circulating blood of a living being, i.e., a human, over a range of shear rates for diagnostics and treatment, such as detecting/reducing blood viscosity, work of the heart, contractility of the heart, for detecting/reducing the surface tension of the blood, for detecting plasma viscosity, for explaining/countering endothelial cell dysfunction, for providing high and low blood vessel wall shear stress data, red blood cell deformability data, lubricity of blood, and for treating different ailments such as peripheral arterial disease in combination with administering to a living being at least one pharmaceutically acceptable agent. Agents pharmaceutically effective to regulate at least one of the aforementioned blood parameters are used to adjust distribution of a substance through the bloodstream. For example, when blood viscosity is a blood flow parameter monitored, an agent is selected from i.v. diluents, red blood cell deformability agents, antiurea agents, oral contraceptives, antidiabetic agents, antiarrhythmics, antihypertensives, antihyperlipidemics, antiplatelet agents, appetite suppressants, antiobesity agents, blood modifiers, smoking deterrent agents, and

nutritional supplements.

2002:185688 HCAPLUS <<LOGINID::20080722>>

DN 136:252567

TI Methods for drug administration and distribution based on monitoring blood viscosity and other parameters for diagnostics and treatment

IN Kensey, Kenneth

PA USA

AN

SO U.S. Pat. Appl. Publ., 46 pp., Cont.-in-part of U.S. Ser. No. 819,924.

CODEN: USXXCO DT Patent

LA English

FAN.		8																	
	PAI	ENT I				KIN		DATE			APPL						ATE		
PI	PI US 20020032149 US 6019735 CA 2301161 WO 9910724					A1 A A1 A2		2002 2000 1999 1999	0314 0201 0304		US 2: US 1: CA 1: WO 1:	001-: 997-: 998-:	8413 9199 2301	89 06 161		1:	9970 9980	424 < 828 < 826 <	<
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			MC,		PT,			CY, BJ,								ML,	MR,	NE,	
	HU 2001000201 HU 2001000201					A2 A3		2001 2004			HU 2	001-	201			1	9980	826 <	:
	JP US	5029 2001 6322	5143: 524	84		A T B1		2001 2001 2001	0911 1127		NZ 1: JP 2: US 1:	000-	5079	94		1:	9980 9991	826 < 826 < 112 <	<
	NO	2000	0009			B1 A A		2001 2000 2001	0225		US 2 NO 2 MX 2	000-	944			2	0000	210 < 225 < 228 <	<
	MX 200002073 US 6428488 WO 2002009583			B1 20020806 US 2000-615340 A2 20020207 WO 2001-US23696						2	0000	712 < 730 <	<						
		2002 W:	0095	83	AT.	A3		2002 AU,	0425										
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			RO, UZ,	RU, VN,	SD, YU,	SE, ZA,	SG, ZW,	MD, SI, SZ,	SK, BE,	SL, CY,	TJ, FR,	TM, GR,	TR, IE,	TT,	TZ,	UA,	UG,	US,	
		2002	0088		CI,	A1		GN, 2002	0711		ML, US 2				TD,		0011	227 <	<
	WO	2002 2002	0797			B2 A2 A3		2003 2002 2003	1010		WO 2	002-	JS39	84		2	0020	207 <	<
	110	W:	AE, CO,	AG, CR,	CU,	AM, CZ,	AT, DE,	AU, DK, IN,	AZ, DM,	DZ,	EC,	EE,	ES,	FI,	GB,	GD,	GE,	GH,	
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		RW:	GH, KZ, IE,	GM, MD, IT,	KE, RU, LU,	LS, TJ, MC,	MW, TM, NL,	MZ, AT, PT,	BE, SE,	CH, TR,	CY,	DE,	DK,	ES,	FI,	FR,	GB,	GR,	
		2002 6571	0184			A1 B2		SN, 2002 2003	1212		US 2	002-	1561	65		2	0020	528 <	:

PRAI	US	1997-919906	A2	19970828	<
	US	1999-439795	A2	19991112	<
	US	2000-501856	A2	20000210	<
	US	2000-628401	A2	20000801	<
	US	2000-727950	A2	20001201	<
	US	2001-819924	A2	20010328	<
	US	1997-966076	A	19971107	<
	WO	1998-US17657	W	19980826	<
	US	2000-615340	A3	20000712	<
	US	2000-228612P	P	20000828	<
	US	2001-789350	B2	20010221	<
	US	2001-828761	A	20010409	<
	US	2001-839785	A	20010420	<
	US	2001-841389	A	20010424	<
	US	2001-897164	A3	20010702	<

- ANSWER 6 OF 8 HCAPLUS COPYRIGHT 2008 ACS on SIN T.9
- ΤI Dietary supplement containing histidine for alleviating dysmenorrhea, endometriosis, and pre-term labor
- AB The present invention relates to methods for alleviating disorders or chronic conditions of the female reproductive system, such as dysmenorrhea, endometrial pain, and pre-term labor, through dietary supplementation with histidine (500 mg-30 g/daily). The invention relates further to novel combination supplements of histidine in conjunction with other nutritional supplement materials which are preferably also useful in alleviating the above-mentioned disorders or conditions. method for administering a dietary histidine supplement in conjunction with one or more sep. formulated therapeutic drugs also known to be useful in treating these female reproductive conditions is also disclosed. For example, a capsule was prepared containing 300 mg of L-histidine, 250 mg CaCO3, and lactose as a carrier.
- AN 2001:240150 HCAPLUS <<LOGINID::20080722>>
- DN 134:271255
- Dietary supplement containing histidine for alleviating dysmenorrhea, endometriosis, and pre-term labor
- TN Peterson, Johnny W.; Thomas, Peter G.
- PA USA
- SO U.S., 16 pp.
- CODEN: USXXAM
- DT Patent
- LA English

DAM ONT 1

PAN.CNI I				
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 6211221	B1	20010403	US 1999-285717	19990405 <
PRAI US 1999-285717		19990405 <	<	
RE.CNT 6 THERE ARE	6 CITED	REFERENCES	AVAILABLE FOR THIS RECO	RD
ALL CITATI	ONS AVA	ILABLE IN TH	HE RE FORMAT	

ANSWER 7 OF 8 HCAPLUS COPYRIGHT 2008 ACS on STN L9

Bright white film coatings and film coating compositions therefor AB A dry film coating composition used to make a bright white film coating for nutritional supplements, pharmaceutical tablets, and the like, comprises dextrose, an auxiliary film-former, and titania. Optionally, but advantageously, the coating composition also may include one or more of the following components: a plasticizer, a surfactant, a flow aid, and a preservative. The composition provides a film coating that possesses good film adhesion and a smooth surface. A coating dispersion was formulated containing dextrose 32, HPMC (Pharmacoat E-50) 10, PEG-8000 8, HPMC (Pharmacoat E-15) 5, Na CMC 6, Na citrate 3, mineral oil 3, titania 32, and Polysorbate-80 1 %. The dispersion was sprayed onto APAP tablets and

this produced a bright white film coating.

1999:77458 HCAPLUS <<LOGINID::20080722>>

DN

AN

Bright white film coatings and film coating compositions therefor

IN Grillo, Susan M.; Korchok, Brian; Kinsey, Bruce; Hartman, Melanie; Porter, Stuart C.; Steffenino, Rita; Reyes, George; Burke, Thomas J. PA Berwind Pharmaceutical Services, Inc., USA

SO PCT Int. Appl., 34 pp.

CODEN: PIXXD2

Patent

LA English FAN CNT 1

PAIN.		TENT I						DATE				ICAT				D	ATE		
PI	WO	9903														1	9980	716	<
		W:						BA,											
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								LR,											
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						VN,													
		RW:						SD,											
								IT,					SE,	BF,	ΒJ,	CF,	CG,	CI,	
			CM,	GΑ,	GN,	GW,	ML,	MR,	ΝE,	SN,	TD,	TG							
	US	6248	391			В1		2001	0619		US 1	997-	8954	84		1	9970	716	<
	CA	2296	5248391 B 2296425 A 2296425 C								CA 1	998-	2296	425		1	9980	716	<
	CA	2296	425			С		2007											
	ΑU	9884	107			A		1999	0210		AU 1	998-	8410	7		1	9980	716	<
		7384																	
	EΡ	1011						2000											
		R:						ES,	FR,	GB,	GR,	IT,	LI,	LU,	NL,	SE,	PT,	ΙE,	
						FI,													
	BR	9811	106			A		2000											
	TR	2000	0012	2		T2		2000											
	ZA	9811 2000 9806 2001	339			A		2000											
	JP	2001	5101	49		T		2001											
		200000570 A							1 MX 2000-570 2000011 1 US 2001-754937 2001010										
		6267										001-	7549	37		2	0010	105	<
PRAI								1997											
		1998						1998											
RE.CI	IΤ	6	TH	ERE 2	ARE	6 CI'	ΓED	REFE	RENC	ES A	VAIL	ABLE	FOR	THI	S RE	CORD			

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 8 OF 8 HCAPLUS COPYRIGHT 2008 ACS on STN

Invasion of tissue culture cells by diarrheagenic strains of Escherichia TI coli which lack the enteroinvasive inv gene

Invasive Escherichia coli strains of certain serotypes invade by the same AB mechanism as the Shigella sp. It has been proposed that invasion of epithelial cells by EPEC strains may also occur; this is a previously overlooked property. In the present study E. coli strains isolated from patients with diarrhea or ulcerative colitis, lacking the inv plasmid mediating classical invasion, but hybridizing with probes for different adhesins, were analyzed for their ability to invade HeLa and Caco-2 cells. The majority of strains invaded Caco-2 cells to a higher extent than HeLa cells. Adhesion to Caco-2 cells was a prerequisite for subsequent invasion of the cells but EAF, eae, EAgg and other known virulence factors were not sufficient to mediate invasion. In 8/9 E. coli strains invasion was enhanced after growth under iron restriction. Growth during anaerobic conditions did not influence subsequent invasion by E. coli strains whereas 6/9 strains had their invasive ability significantly decreased after growth in the presence of 1% glucose. The invasive process was inhibited by mannose but not by

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lactose, fucose or galactose. The data indicate that strains of
     E. coli may invade Caco-2 cells by novel mechanisms which require
     adhesion to the cells but which differ from those of Salmonella
     sp., Yersinia sp., Shigella sp. and classical enteroinvasive E. coli.
    1996:347747 HCAPLUS <<LOGINID::20080722>>
AN
DN
     125:83399
OREF 125:15695a,15698a
     Invasion of tissue culture cells by diarrheagenic strains of Escherichia
     coli which lack the enteroinvasive inv gene
AU
     Gevid, Aberra; Fletcher, Jon; Gashe, Brehanu A.; Ljungh, Asa
CS
     Department of Medical Microbiology, University of Lund, Soelvegatan 23,
     Lund, S-223 62, Swed.
SO
     FEMS Immunology and Medical Microbiology (1996), 14(1), 15-24
     CODEN: FIMIEV; ISSN: 0928-8244
PB
    Elsevier
DT
    Journal
LA
    English
=> d his
     (FILE 'HOME' ENTERED AT 13:29:10 ON 22 JUL 2008)
     FILE 'HCAPLUS' ENTERED AT 13:30:24 ON 22 JUL 2008
           3287 S URSODEOXYCHOL?
1.2
         139310 S ISCHEM? OR STROKE OR NEUROPROTECTIVE
T.3
             28 S L1 AND L2
L4
             19 S L3 AND (PY<2005 OR AY<2005 OR PRY<2005)
     FILE 'STNGUIDE' ENTERED AT 13:30:30 ON 22 JUL 2008
     FILE 'HCAPLUS' ENTERED AT 13:30:43 ON 22 JUL 2008
     FILE 'STNGUIDE' ENTERED AT 13:30:46 ON 22 JUL 2008
    FILE 'HCAPLUS' ENTERED AT 15:30:51 ON 22 JUL 2008
L5
          96359 S PREBIOTIC OR ENTERAL OR DIARRHEA OR NUTRITIONAL
L6
         493913 S ADHESIVE OR ADHESION
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     FILE 'HCAPLUS' ENTERED AT 15:31:22 ON 22 JUL 2008
L7
          95742 S ((ARABINO OR MANNO OR GALACTO OR ISOMALTO OR SIALYL) (W)OLIGOS
L8
             19 S L5 AND L6 AND L7
     FILE 'STNGUIDE' ENTERED AT 15:31:26 ON 22 JUL 2008
     FILE 'HCAPLUS' ENTERED AT 15:32:11 ON 22 JUL 2008
L9
              8 S L8 AND (PY<2004 OR AY<2004 OR PRY<2004)
     FILE 'STNGUIDE' ENTERED AT 15:32:20 ON 22 JUL 2008
     FILE 'HCAPLUS' ENTERED AT 15:32:30 ON 22 JUL 2008
     FILE 'STNGUIDE' ENTERED AT 15:32:31 ON 22 JUL 2008
=> log hold
COST IN U.S. DOLLARS
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                                                                 95.85
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CA SUBSCRIBER PRICE 0.00 -21.60

SESSION WILL BE HELD FOR 120 MINUTES STN INTERNATIONAL SESSION SUSPENDED AT 15:32:35 ON 22 JUL 2008

Connecting via Winsock to STN

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LOGINID:SSPTAEX01623

PASSWORD:

* * * * * RECONNECTED TO STN INTERNATIONAL * * * * * * SESSION RESUMED IN FILE 'STNGUIDE' AT 15:49:38 ON 22 JUL 2008 FILE 'STNGUIDE' ENTERED AT 15:49:38 ON 22 JUL 2008 COPYRIGHT (C) 2008 AMERICAN CHEMICAL SOCIETY (ACS)

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=> file hcaplus COST IN U.S. DOLLARS	SINCE FILE	TOTAL SESSION
FULL ESTIMATED COST	ENTRY 0.12	95.91
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-21.60

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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4 FILE LAST UPDATED: 21 Jul 2008 (20080721/ED)

HCAplus now includes complete International Patent Classification (IPC) reclassification data for the second quarter of 2008.

New CAS Information Use Policies, enter HELP USAGETERMS for details.

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s HT29

L10 2473 HT29

=> s 15 and 16 and 110

L11 1 L5 AND L6 AND L10

=> s 15 and 17 and 110

L12 1 L5 AND L7 AND L10

=> s 111 and (PY<2004 or AY<2004 or PRY<2004)

23986246 PY<2004 4779965 AY<2004

4250851 PRY<2004

L13 0 L11 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> s 112 and (PY<2004 or AY<2004 or PRY<2004)

23986246 PY<2004 4779965 AY<2004

4250851 PRY<2004

L14 0 L12 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnguide

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FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: Jul 18, 2008 (20080718/UP).

=> d 111 ti abs bib

CA SUBSCRIBER PRICE

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

- L11 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Investigation of the biological activities of pectic oligosaccharides using in vitro models of the human colon
- AB Oligosaccharides derived from pectins by either enzymic hydrolysis or by flash extraction were investigated for their prebiotic activities and for their ability to prevent the adhesion of pathogenic bacteria

and bacterial toxins. Potential prebiotic activity was evaluated using pH-controlled batch cultures inoculated with human faecal samples. Microbial population changes were monitored by fluorescent in situ hybridization techniques. Pectic oligosaccharides derived from either manufacturing route displayed potential prebiotic properties in that they selectively increased the populations of beneficial bacteria such as bifidobacteria and lactobacilli and decreased undesirable bacteria such as clostridia. The oligosaccharides had a more selective fermentation

than

the parent polysaccharides. Antiadhesive activity was evaluated using the colon cancer cell line, HT29. Pectic oligosaccharides displayed some degree of protection against Escherichia coli Shiga-like toxins. In addition, pectic oligosaccharides derived by flash extraction from citrus

wastes

displayed antiadhesive activity against enteropathogenic and verotoxigenic strains of E. coli. During the execution of this work, we have also discovered preliminary data suggesting that pectic oligosaccharides may act to induce apoptosis in the colon cancer line used.

AN

- 2005:186972 HCAPLUS <<LOGINID::20080722>> ΤI
- Investigation of the biological activities of pectic oligosaccharides using in vitro models of the human colon
- Rastall, Robert A.; Manderson, Kirstie; Hotchkiss, Arland T.; Gibson, Glenn R. CS School of Food Biosciences, The University of Reading, Reading, RG6 6AP,
- SO
- Abstracts of Papers, 229th ACS National Meeting, San Diego, CA, United States, March 13-17, 2005 (2005), CELL-147 Publisher: American Chemical Society, Washington, D. C. CODEN: 69GQMP
- DT Conference; Meeting Abstract
- LA English

=> d 112 ti

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:v

- L12 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2008 ACS on STN
- A novel galactooligosaccharide mixture increases the bifidobacterial population numbers in a continuous in vitro fermentation system and in the proximal colonic contents of pigs in vivo

=> d 112 ti abs bib

YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:v

- L12 ANSWER 1 OF 1 HCAPLUS COPYRIGHT 2008 ACS on STN
- A novel galactooligosaccharide mixture increases the bifidobacterial population numbers in a continuous in vitro fermentation system and in the proximal colonic contents of pigs in vivo
- Prebiotics are nondigestible food ingredients that encourage proliferation of selected groups of the colonic microflora, thereby altering the composition toward a more beneficial community. In the present study, the prebiotic potential of a novel galactooligosaccharide (GOS) mixture, produced by the activity of galactosyltransferases from Bifidobacterium bifidum 41171 on lactose, was assessed in vitro

and in a parallel continuous randomized pig trial. In situ fluorescent hybridization with 165 rRNA-targeted probes was used to investigate changes in total bacteria, bifidobacteria, lactobacilli, bacteroides, and Clostridium histolyticum group in response to supplementing the novel GOS mixture In a 3-stage continuous culture system, the bifidobacterial nos. for the first 2 vessels, which represented the proximal and traverse colon, increased (P < 0.05) after the addition of the oligosaccharide mixture In addition, the oligosaccharide mixture strongly inhibited the attachment of enterohepatic Escherichia coil (P < 0.01) and Salmonella enterica serotype Typhimurium (P < 0.01) to HT29 cells. Addition of the novel mixture at 4% (Wt:wt) to a com. diet increased the d. of bifidobacteria (P < 0.001) and the acetate concentration (P < 0.001), and decreased the pH (P < 0.001) compared with the control diet and the control diet supplemented with inulin, suggesting a great prebiotic potential for the novel oligosaccharide mixture

- AN 2005:628823 HCAPLUS <<LOGINID::20080722>>
- TI A novel galactooligosaccharide mixture increases the bifidobacterial population numbers in a continuous in vitro fermentation system and in the proximal colonic contents of pigs in vivo
- AU Tzortzis, George; Goulas, Athanasios K.; Gee, Jennifer M.; Gibson, Glenn R.
- ${\tt CS} \quad {\tt School} \ \, {\tt of} \ \, {\tt Food} \ \, {\tt Biosciences}, \ \, {\tt The} \ \, {\tt University} \ \, {\tt of} \ \, {\tt Reading}, \ \, {\tt Reading}, \ \, {\tt RG6} \ \, {\tt 6AP}, \\ {\tt UK} \quad \, {\tt UK}$
- SO Journal of Nutrition (2005), 135(7), 1726-1731 CODEN: JONUAI; ISSN: 0022-3166
- PB American Society for Nutritional Sciences
- DT Journal
- LA English
- RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> file hcaplus COST IN U.S. DOLLARS FULL ESTIMATED COST	SINCE FILE ENTRY 0.18	TOTAL SESSION 113.21
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION -23.20

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FILE COVERS 1907 - 22 Jul 2008 VOL 149 ISS 4 FILE LAST UPDATED: 21 Jul 2008 (20080721/ED)

HCAplus now includes complete International Patent Classification (IPC)

reclassification data for the second quarter of 2008.

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This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s 15 and 110

L15 26 L5 AND L10

=> s 16 and 110

L16 188 L6 AND L10

=> s 115 and (PY<2004 OR AY<2004 OR PRY<2004)

23986246 PY<2004 4779965 AY<2004 4250851 PRY<2004

L17 15 L15 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> s 116 AND (PY<2004 OR AY<2004 OR PRY<2004)

23986246 PY<2004 4779965 AY<2004 4250851 PRY<2004

115 L16 AND (PY<2004 OR AY<2004 OR PRY<2004)

=> file stnguide

FILE 'STNGUIDE' ENTERED AT 15:53:29 ON 22 JUL 2008 USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT COPYRIGHT (C) 2008 AMERICAN CHEMICAL SOCIETY (ACS)

FILE CONTAINS CURRENT INFORMATION.

LAST RELOADED: Jul 18, 2008 (20080718/UP).

=> d 117 1-15 ti abs bib YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:y

- L17 ANSWER 1 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Method of augmenting the antitumor activity of anticancer agents by selenium compounds
- AB A method for augmenting the antitumor activity of anti-cancer agents is provided. The method comprises administering to an individual an anti-cancer agent and a selenium compound A method is also provided for inhibiting the growth of a tumor which has proven to be refractory to anticancer agents. The methods comprises administration of selenium compound followed by administration of the anticancer agent.

- AN 2005:983780 HCAPLUS <<LOGINID::20080722>>
- DN 143:222496
- Method of augmenting the antitumor activity of anticancer agents by TT selenium compounds
- TM Fakih, Marwan; Rustum, Youcef M.; Pendvala, Lakshmi; Smith, Patrick PA
- SO U.S. Pat. Appl. Publ., 24 pp., Cont.-in-part of U.S. Ser. No. 844,800. CODEN: USXXCO
- Patent LA English
- FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 20050197399	A1	20050908	US 2005-79633	20050311 <
	US 20050026852	A1	20050203	US 2004-844800	20040513 <
	US 20060258697	A1	20061116	US 2006-405377	20060417 <
PRAI	US 2003-471183P	P	20030513	<	
	US 2004-844800	A2	20040513		
	US 2005-79633	B2	20050311		

- L17 ANSWER 2 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- Serotypes, virulence factors, antibiotic sensitivity, beta-lactamase activity and plasmid analysis of Salmonella from children with diarrhea in Tripoli (Libva)
- A total of 21 Salmonella strains isolated in Libya (16 from children with diarrhea and 5 from healthy controls) were serotyped and studied for their cell invasive ability, production of cytotoxin, antibiotic susceptibility, β -lactamase activity and plasmid profiles. Eight different serotypes of Salmonella were identified: 6 S. saintpaul, 4 S. wien (1 from control), 2 S. newport, 2 S. muenchen (1 from control), 2 S. typhimurium (1 from control), 2 S. hadar (1 from control), 2 S. reading (1 from control), 1 S. kottbus. Twenty (95%) were pos. in the invasiveness assay using HeLa cells, and all (100%) were neg. for cytotoxin production in HT29 cells. More than 40% were resistant to ampicillin, cefalexin, cefamandole, cefoperazone, chloramphenicol, gentamicin, mezlocillin and trimethoprim-sulfamethoxazole and 100% were susceptible to the new quinolones. Most (67%) of the strains harbored plasmids and 43% produced β-lactamase. A strong association was observed between the presence of more than one plasmid, β-lactamase activity, and multiple-resistance to antimicrobial agents and serotypes S. saintpaul and S. wien. Curing expts. with acridine orange showed that 2 plasmids (33 and 1.4 megadaltons) might be responsible for the resistance to chloramphenicol and gentamicin. The present study demonstrated that multiple-resistant salmonellae are widespread in Libya and the resistance is mainly plasmid mediated.
- AN 2003:100203 HCAPLUS <<LOGINID::20080722>>
- DN 138:300365
- TI Serotypes, virulence factors, antibiotic sensitivity, beta-lactamase activity and plasmid analysis of Salmonella from children with diarrhea in Tripoli (Libya)
- El-Ghodban, A.; Ghenghesh, K. S.; Marialigeti, K.; Abeid, S. ΑU
- CS Department of Microbiology, Eotvos Lorand University, Budapest, H-1117, Hung.
- Acta Microbiologica et Immunologica Hungarica (2002), 49(4), 433-444 CODEN: AMIHEF; ISSN: 1217-8950
- PB Akademiai Kiado
- DT Journal
- English
- RE.CNT 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

- L17 ANSWER 3 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Human serum amyloid A3 peptide enhances intestinal MUC3 expression and inhibits EPEC adherence
- ${\tt AB}$ We previously determined that the N-terminal region of bovine mammary-associated

serum amyloid A3 (M-SAA3) increased intestinal mucin MUC3 levels in HT29 human intestinal cells by .apprx.2.5-fold, relative to untreated cells. This study shows that the human M-SAA3 N-terminal peptide further enhances MUC3 transcript levels by .apprx.4.3-fold in these cells (p<0.02), implicating a species-specific interaction. Furthermore, immunofluorescence and immunoblot anal. using a MUC3-specific monoclonal antibody confirms that the human M-SAA3 peptide stimulates MUC3 protein expression and secretion by the HT29 cells. More importantly, pretreatment of the cells with the peptide causes a subsequent 73% decrease in the adherence of enteropathogenic Escherichia coli (EPEC) to these cells, relative to untreated cells (p <0.01). The intestinal mucin MUC3 has been shown to provide a protective barrier in the gut and inhibit adherence of pathogens to the gut wall. Therefore, a means to increase MUC3 protein expression by a colostrum-associated peptide or protein may be a highly effective prophylactic treatment for the prevention of gastrointestinal diseases such as necrotizing enterocolitis and infectious diarrhea.

- AN 2002:972020 HCAPLUS <<LOGINID::20080722>>
- DN 139:78878
- TI Human serum amyloid A3 peptide enhances intestinal MUC3 expression and inhibits EPEC adherence
- AU Larson, Marilynn A.; Wei, Shu H.; Weber, Annika; Mack, David R.; McDonald, Thomas L.
- CS Department of Pathology and Microbiology, University of Nebraska Medical Center, Omaha, NE, 68198, USA
- SO Biochemical and Biophysical Research Communications (2003), 300(2), 531-540
 - CODEN: BBRCA9; ISSN: 0006-291X
- PB Elsevier Science
- DT Journal
- LA English
- RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L17 ANSWER 4 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Enteroinvasive bacteria alter barrier and transport properties of human intestinal epithelium: role of iNOS and COX-2
- AB Various invasive pathogens cause diarrhea, but the mechanism(s) are poorly understood. We hypothesized that nitric oxide and prostaglandins might modulate chloride secretory and barrier properties of the infected intestinal epithelium and that diarrhea is caused, in part, by altered expression of inducible NO synthase (iNOS) and cyclooxygenase 2 (COX-2). Studies were conducted in human intestinal epithelial cell lines (HT29/cl.19A, Caco-2, and T84). Cells were infected with enteroinvasive Escherichia coli (EIEC 029:NM) or Salmonella dublin (SD), or nonpathogenic, noninvasive bacteria (Streptococcus thermophilus [ST] and Lactobacillus acidophilus [LA]). Infected cells and controls were tested for transepithelial resistance, chloride secretion, prostaglandin E2, guanosine 3',5'-cyclic monophosphate and adenosine 3',5'-cyclic monophosphate, and protein expression. Cells infected with EIEC or SD, but not uninfected controls or ST/LA-exposed monolayers, showed a progressive reduction in transepithelial resistance starting at 6-12 h. Infected HT29/c1.19A and Caco-2 cells, but not T84 cells, also showed an increase in total nitrite. Expression of iNOS, and consequently COX-2, was also increased, followed by increased

production of proetaglandine and cyclic nucleotides. Furthermore, basal and stimulated chloride secretory responses to various agonists were enhanced in HT29/cl.19A and Caco-2 cells after infection with enteroinvasive bacteria, and this effect was reversed for some agonists by iNOS or CCV-2 inhibitors. Increased expression of cystic fibrosis transmembrane conductance regulator and NKCCl was also observed in EIEC or SD-infected cells vs. controls, secondary to NO synthase activity. Conclusions: Up-regulation of iNOS and COX-2 by enteroinvasive bacteria can modulate chloride secretion and barrier function in intestinal epithelial cells. Thus, these enzymes represent possible therapeutic targets in infectious diarrhes.

AN 2002:317594 HCAPLUS <<LOGINID::20080722>>

DN 137:199457

TI Enteroinvasive bacteria alter barrier and transport properties of human intestinal epithelium: role of iNOS and COX-2

AU Resta-Lenert, Silvia; Barrett, Kim E.

CS Department of Medicine, University of California, San Diego, School of Medicine, San Diego, CA, USA SO Gastroenterology (2002), 122(4), 1070-1087

CODEN: GASTAB; ISSN: 0016-5085

PB W. B. Saunders Co.

DT Journal LA English

- RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L17 ANSWER 5 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI A functional NSP4 enterotoxin peptide secreted from rotavirus-infected cells
- AB Previous studies have shown that the nonstructural glycoprotein NSP4 plays a role in rotavirus pathogenesis by functioning as an enterotoxin. One prediction of the mechanism of action of this enterotoxin was that it is secreted from virus-infected cells. In this study, the media of cultured (i) insect cells infected with a recombinant baculovirus expressing NSP4, (ii) monkey kidney (MA104) cells infected with the simian (SA11) or porcine attenuated (OSU-a) rotavirus, and (iii) human intestinal (HT29) cells infected with SA11 were examined to determine if NSP4 was detectable. Sodium dodecyl sulfate-PAGE-Western blotting, immunopptn. and N-terminal amino acid sequencing identified, in the early media from virus-infected cells, a secreted, cleavage product of NSP4 with an apparent mol. weight of 7,000 that represented amino acids 112 to 175 (NSP4 aa112-175). The secretion of NSP4 aa112-175 was not affected by treatment of cells with brefeldin A but was abolished by treatment with nocodazole and cytochalasin D, indicating that secretion of this protein occurs via a nonclassical, Golgi apparatus-independent mechanism that utilizes the microtubule and actin microfilament network. A partial gene fragment coding for NSP4 aa112-175 was cloned and expressed using the baculovirus-insect cell system. Purified NSP4 aa112-175 increased intracellular calcium mobilization in intestinal cells when added exogenously, and in insect cells when expressed endogenously, similar to full-length NSP4. NSP4 aa112-175 caused diarrhea in neonatal mice, as did full-length NSP4. These results indicate that NSP4 aa112-175 is a functional NSP4 enterotoxin peptide secreted from rotavirus-infected cells.
- AN 2001:205105 HCAPLUS <<LOGINID::20080722>>
- DN 135:3857
- ${\tt TI} \quad {\tt A} \ {\tt functional} \ {\tt NSP4} \ {\tt enterotoxin} \ {\tt peptide} \ {\tt secreted} \ {\tt from} \ {\tt rotavirus-infected} \ {\tt cells}$
- AU Zhang, Mingdong; Zeng, Carl Q.-Y.; Morris, Andrew P.; Estes, Mark K. CS Division of Molecular Virology, Baylor College of Medicine, Houston, TX, 77030, USA

- SO Journal of Virology (2000), 74(24), 11663-11670 CODEN: JOVIAM; ISSN: 0022-538X
- PB American Society for Microbiology
- DT Journal
- LA English
- RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L17 ANSWER 6 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Inter- and intra-individual variation of fecal water - genotoxicity in human colon cells
 - Exogenous nutritional factors modulate the fecal contents leading to an enhanced or reduced burden with toxic and cancerogenic factors. These factors are thought to contribute to colon cancer by inducing mutations or enhancing proliferation in colon cells. Fecal water more or less causes these effects in model systems and thus could be the basis for valuable biomarker approaches. Our investigations are aimed at determining geno- and cytotoxicity of fecal water in human colon cell lines in vitro. We are developing techniques for their applicability as biomarker tests during dietary intervention studies. Fecal water is isolated by centrifugation of the feces at 25 000+q and added to cultured human colon cells (HT29). Membrane damage as assessed by trypan blue exclusion is determined as a measure for cytotoxicity. Semiguant, anal, of inducible DNA damage (breaks and alkali labile sites) are analyzed with the single cell microgelelectrophoresis assay (comet-assay) and oxidized DNA bases by the addnl. use of repair specific enzymes. We have now determined baseline toxic activities and calculated inter- and intra-individual and -exptl. coeffs. of variation for fecal water from different subjects consuming similar or different diets. Most fecal water induced DNA damage and oxidized DNA bases in HT29 clone 19a cells (0.9-9.14 fold and 1.7-4.9 fold, resp. in comparison to the NaCl controls). Intra- and inter-exptl. coeffs. (CV) of variation, were in a similar order of magnitude and ranged from 6.9 to 31.4. In contrast both intra- and inter-individual variability were considerably higher (CV-ranges of 29.7-76.6 and 21.3-64.0, resp.). Interestingly, these inter-individual values were not lowered when subjects consumed identical diets (CV-ranges of 28.4-126.0). However, following intervention with certain protective dietary regimens (e.g. lignan containing bread) significant redns. of fecal water-induced genotoxicity can be observed Therefore, in spite of the expected and observed degrees of variation in this methodol., effective exptl. protocols may still lead to detectable modulations of the level of toxic and genotoxic effects.
- AN 2000:875450 HCAPLUS <<LOGINID::20080722>>
- DN 134:158721
- TΙ Inter- and intra-individual variation of fecal water - genotoxicity in human colon cells
- AII Osswald, K.; Becker, T. W.; Grimm, M.; Jahreis, G.; Pool-Zobel, B. L. CS Institute for Nutrition, Department of Nutritional Toxicology,
- Friedrich-Schiller University, Jena, D-07743, Germany
- Mutation Research, Genetic Toxicology and Environmental Mutagenesis (SO 2000), 472(1-2), 59-70 CODEN: MRGMFI; ISSN: 1383-5718
- PB Elsevier B.V.
- DT Journal
- LA English
- RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L17 ANSWER 7 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- Application of confocal laser scanning microscopy to detect oxidative stress in human colon cells

AB Introduction - Excess of intracellular reactive oxygen species in relation to antioxidative systems results in an oxidative environment which may modulate gene expression or damage cellular mols. These events are expected to greatly contribute to processes of carcinogenesis. Only few studies are available on the oxidative/reductive conditions in the colon, an important tumor target tissue. It was the objective of this work to further develop methods to assess intracellular oxidative stress within human colon cells as a tool to study such assocns. in nutritional toxicol. Methods - We have measured H2O2-induced oxidative stress in different colon cell lines, in freshly isolated human colon crypts, and, for comparative purposes, in NIH3T3 mouse embryo fibroblasts. Detection was performed by loading the cells with the fluorigenic peroxide-sensitive dye 6-carboxy-2',7'-dichlorodihydrofluorescein diacetate (diacetoxymethyl ester), followed by in vitro treatment with H2O2 and fluorescence detection with confocal laser scanning microscopy (CLSM). Using the microgel electrophoresis ("Comet") Assay, we also examined HT29 stem and clone 19A cells and freshly isolated primary colon cells for their relative sensitivity toward H2O2-induced DNA damage and for steady-state levels of endogenous oxidative DNA damage. Results A dose-response relationship was found for the H2O2-induced dye decomposition in NIH3T3 cells (7.8-125 µM H2O2) whereas no effect occurred in the human colon tumor cell lines HT29 stem and HT29 clone 19A (62-1000 uM H2O2). Fluorescence was significantly increased at 62 uM H2O2 in the human colon adenocarcinoma cell line Caco-2. In isolated human colon crypts, the lower crypt cells (targets of colon cancer) were more sensitive towards H2O2 than the more differentiated upper crypt cells. In contrast to the CLSM results, oxidative DNA damage was detected in both cell lines using the Comet Assay. Endogenous oxidative DNA damage was highest in HT29 clone 19A, followed by the primary colon cells and HT29 stem cells. Conclusions Oxidative stress in colon cells leads to damage of macromols. which is sensitively detected in the Comet Assay. The lacking response of the CLSM-approach in colon tumor cells is probably due to intrinsic modes of protective activities of these cells. In general, however, the CLSM method is a sensitive technique to detect very low concns. of H2O2-induced oxidative stress in NIH3T3 cells. Moreover, by using colon crypts it provides the unique possibility of assessing cell specific levels of oxidative stress in explanted human tissues. Our results demonstrate that the actual target cells of colon cancer induction are indeed susceptible to the oxidative activity of H2O2.

AN 2000:763779 HCAPLUS <<LOGINID::20080722>>

DN 134:53301

TI Application of confocal laser scanning microscopy to detect oxidative stress in human colon cells
AU Liegibel, Ute M.; Abrahamse, Salomon L.; Pool-Zobel, Beatrice L.;

Rechkemmer, Gerhard
CS Department of Nutritional Toxicology, Institute for Nutrition,

Friedrich-Schiller-University, Jena, 07743, Germany
SO Free Radical Research (2000), 32(6), 535-547

CODEN: FRARER; ISSN: 1071-5762

PB Harwood Academic Publishers

DT Journal

LA English

RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 8 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN

TI The in vitro manipulation of carbohydrate metabolism: a new strategy for deciphering the cellular defence mechanisms against nitric oxide attack

AB This study was aimed at examining the effects of manipulating the carbohydrate source of the culture medium on the cellular sensitivity of

epithelial cells to an oxidative attack. Our rationale was that substituting galactose for glucose in culture media would remove the protection afforded by glucose utilization in two major metabolic pathways, i.e. anaerobic glycolysis and/or the pentose phosphate pathway (PPP), which builds up cellular reducing power. Indeed, we show that the polarized human colonic epithelial cell line HT29-C1.16E was sensitive to the deleterious effects of the NO donor PAPANONOate [3-(2-hydroxy-2-nitroso-1-propylhydrazino)-1-propanamine] only in galactose-containing medium. In such medium NO attack led to cytotoxic and apoptotic cell death, associated with formation of derivs, of NO auto-oxidation (collectively termed NOx) and peroxynitrite, leading to intracellular GSH depletion and nitrotyrosine formation. The addition of 2-deoxyglucose, a non-glycolytic substrate, to galactose-fed cells protected HT29 -C1.16E cells from NO attack and maintained control GSH levels through its metabolic utilization in the PPP, as shown by 14CO2 production from 2-deoxy[1-14C]glucose. Therefore, increasing the availability of reducing equivalent without interfering with energy metabolism is able to prevent NO-induced cell injury. Finally, this background provides the conceptual framework for establishing nutritional manipulation of cellular metabolic pathways that could provide new means for (i) deciphering the mechanisms of cell injury by reactive nitrogen species and reactive oxygen species at the whole-cell level and (ii) establishing the hierarchy of intracellular defense mechanisms against these attacks.

- AN 2000:27432 HCAPLUS <<LOGINID::20080722>>
- DN 132:178542
- TI The in vitro manipulation of carbohydrate metabolism: a new strategy for deciphering the cellular defence mechanisms against nitric oxide attack
- AU Le Goffe, Claire; Vallette, Genevieve; Jarry, Anne; Bou-Hanna, Chantal; Laboisse, Christian L.
- CS INSERM CJF 94-04, Faculte de Medecine, Nantes, 44035, Fr.
- SO Biochemical Journal (1999), 344(3), 643-648
- CODEN: BIJOAK; ISSN: 0264-6021 PB Portland Press Ltd.
- DT Journal
- LA English
- RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L17 ANSWER 9 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Rapid and sensitive assay for detection of enterotoxigenic Bacteroides fracilis
- Bacteroides fragilis is an obligatory anaerobic, gram-neg, bacterium found among the normal intestinal flora of humans. Enterotoxigenic strains of B. fragilis (ETBF) have been associated with diarrheal diseases in humans and animals. The enterotoxin of ETBF induces fluid changes in ligated intestinal segments and cytotoxic response in HT29/C1 cells. By using a pair of monoclonal antibodies (MAbs; MAb C3 and MAb 4H8) specific for the lipopolysaccharide of B. fragilis, an assay based on immunomagnetic separation (IMS) in combination with PCR (IMS-PCR) was developed. After DNA extraction, a 294-bp fragment was amplified. The specificity of the IMS-PCR assay was evaluated by adding previously isolated and confirmed ETBF strains to normal fecal samples. All fecal samples to which ETBF strains were added were pos., showing a 100% specificity. The spiked fecal samples were also used for evaluation of the sensitivity of the assay. The detection limit was found to be .apprx.50 CFU/q of feces. By this method 10 clin. fecal samples (5 from patients with diarrhea and 5 from healthy controls) were examined The results of PCR were in accordance with the results of the HT29 /Cl cell assay for all samples. The min. time to retrieval of the final result by the IMS-PCR method is 36 h. The proposed IMS-PCR assay is rapid and sensitive for the direct detection of ETBF in stool samples.

- AN 1998:787491 HCAPLUS <<LOGINID::20080722>>
- DN 130:179434
- Rapid and sensitive assay for detection of enterotoxigenic Bacteroides fragilis
- AU Zhang, Guangming; Weintraub, Andrej
- CS Department of Immunology, Microbiology, Pathology and Infectious Diseases, Division of Clinical and Oral Bacteriology, Karolinska Institute, Huddinge University Hospital, Huddinge, S-141 86, Swed.
- SO Journal of Clinical Microbiology (1998), 36(12), 3545-3548 CODEN: JCMIDW; ISSN: 0095-1137
- PB American Society for Microbiology
- DT Journal
- LA English
- RE.CNT 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L17 ANSWER 10 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Osmotic changes and ethanol modify TFF gene expression in gastrointestinal cell lines
- AB The gastrointestinal tract is exposed to environmental insult as a result of food intake or in pathol. conditions such as diarrhea, and is therefore protected by the mucus layer. As part of it, trefoil factor family peptides (TFFs) are able to modify the viscoelastic properties of the mucus, protect against exptl. ulceration, and promote repair of the epithelia. The authors investigated, using transient reporter gene assays and RT-PCR in the gastric carcinoma cell line MKN45 and colon carcinoma cell lines LS174T and HT29, whether ethanol and osmotic changes can modify transcriptional activity of TFFs. In a mild hypotonic environment (200 mosmol/L) all three TFF genes were up-regulated by at least a factor of 2. In hypertonic medium (400 mosmol/1), TFF1 and TFF3 were down-regulated, whereas TFF2 was up-regulated by elevated concns. of sodium or chloride in MKN45. Raising the osmolality by ethanol resulted in an up-regulation of TFF3 in both colon cell lines but not in the gastric cell line. The authors conclude that alteration in TFF gene expression is a response of gut epithelia to deal with osmotic forces and ethanol.
 - 1998:766907 HCAPLUS <<LOGINID::20080722>>
- DN 130:91534

AN

- TI Osmotic changes and ethanol modify TFF gene expression in gastrointestinal cell lines
- AU Ludeking, Alexander; Fegert, Petra; Blin, Nikolaus; Gott, Peter
- CS Department of Anthropology and Human Genetics, Division of Molecular Genetics, University of Tubingen, Tubingen, D-72074, Germany
- SO FEBS Letters (1998), 439(1,2), 180-184 CODEN: FEBLAL; ISSN: 0014-5793
- PB Elsevier Science B.V.
- DT Journal
- LA English
- RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L17 ANSWER 11 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI In vitro inhibition of Cryptosporidium parvum infection by human monoclonal antibodies
- AB Cryptosporidium parvum infection of the small epithelial intestine causes unremitting diarrhea and malabsorption that can lead to chronic and sometimes fatal illness in patients with AIDS. The illness may be ameliorated by passive oral Ig therapy. The objective of this study was to produce anti-Cryptosporidium human monoclonal antibodies for evaluation as potential therapy. All human monoclonal cell lines that produced C. parvum antibodies were originally generated from the peripheral blood

lymphocytes of a human immunodeficiency virus-seroneq, woman. She had recovered from C. parvum infection and had a high specific antibody titer. Hybridization of these lymphocytes with a tumor cell line was accomplished by hypo-osmolar electrofusion. Twelve clones were identified by ELISA as secreting anti-Cryptosporidium antibodies after the initial hybridization. From the 12 pos. clones, two high antibody-secreting clones, 17A and 17B, were maintained in long-term culture. A second hybridization produced two other human monoclonal cell lines, EC5 and BB2. Human monoclonal antibody from the first two cell lines bound to C. parvum sporozoites and oocvsts by immunofluorescence. The ability of human monoclonal antibodies to inhibit C. parvum infection in vitro was assessed by using a human enterocyte cell line, HT29.74. The antibodies of the four different human hybridomas inhibited infection by 35 to 68% compared to a control irrelevant human monoclonal antibody derived in a similar fashion. Human monoclonal antibodies are candidate mols. for immunotherapy of C. parvum infection.

AN 1997:596458 HCAPLUS <<LOGINID::20080722>>

DN 127:277063

OREF 127:54105a,54108a

TI In vitro inhibition of Cryptosporidium parvum infection by human monoclonal antibodies

AU Elliot, Bethany C.; Wienewski, Adam V.; Johnson, Joan; Fenwick-Smith, Daniela; Wiest, Peter; Hamer, David; Kresina, Thomas; Flanigan, Timothy P. CS Miriam Hospital, Brown University, Providence, RI, 02906, USA

SO Infection and Immunity (1997), 65(9), 3933-3935 CODEN: INFIBR; ISSN: 0019-9567

PB American Society for Microbiology

DT Journal

LA English

RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L17 ANSWER 12 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Bacteroides fragilis toxin rearranges the actin cytoskeleton of HT29/C1 cells without direct proteolysis of actin or decrease in F-actin content

AB Enterotoxigenic strains of B. fragilis associated with childhood diarrhea produce a 20 kDa zinc metalloprotease toxin (BFT). BFT is reported to cleave G-actin in vitro and also causes dramatic rounding and rearrangement of the F-actin cytoskeleton in human intestinal epithelial cell lines (HT29 and HT29/C1). To test the hypothesis that the proteolysis of cellular actin by BFT in vivo may contribute to these alterations in morphol, and cytoskeletal architecture, the authors assessed the F-actin content and the arrangement of the F- and G-actin cytoskeleton in BFT-treated HT29/C1 cells by spectrofluorimetry, confocal microscopy, and immunoblotting. BFT-treated cells were compared to cells treated with C. difficile toxin A (CDA) or cytochalasin D. Using spectrofluorometric quantification, the F-actin content of BFT- and cytochalasin D-treated cells was unchanged in contrast to a significant decrease in CDA-treated cells. By confocal microscopy, the arrangement of F- and G-actin in all treated cells was markedly different than control cells. There was no change in the immunoblotting pattern of actin in the Triton-soluble or -insol. cellular fractions of BFT-treated HT29/C1 cells. Evidently, BFT alters the F- and G-actin cytoskeletal architecture of HT29/Cl cells without direct proteolysis of actin or decrease in F-actin content.

AN 1997:408968 HCAPLUS <<LOGINID::20080722>>

DN 127:46295

OREF 127:8727a,8730a

TI Bacteroides fragilis toxin rearranges the actin cytoskeleton of HT29/C1 cells without direct proteolysis of actin or decrease in

F-actin content

- AU Saidi, Roxan F.; Jaeger, Kristin; Montrose, Marshall H.; Wu, Shaoguang; Sears, Cynthia L.
- CS Division of Gastroenterology, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, MD, 21205, USA
- SO Cell Motility and the Cytoskeleton (1997), 37(2), 159-165
- CODEN: CMCYEO; ISSN: 0886-1544
- PB Wiley-Liss
- DT Journal LA English
- L17 ANSWER 13 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- ${\tt TI}$ Bacteroides fragilis toxin rapidly intoxicates human intestinal epithelial cells (HT29/C1) in vitro
- AB Enterotoxiqenic Bacteroides fragilis strains associated with childhood diarrhea produce a 20-kDa protein toxin (BFT). Purified BFT causes striking morphol. changes in subconfluent human colonic epithelial cells (HT29/C1). In a 3-h HT29/C1 cell assay, the estimated half-maximal effective concentration of BFT was 12.5 pM, and morphol. effects were detectable as early as 30 min and nearly complete by 1.5 h. Concns. as low as 0.5 pM could also cause intoxication, but morphol. changes were detectable only when the assay was extended to 18 h. The onset of this intoxication was concentration dependent and rapid, occurring within minutes (<7 min at 0.25 nM, <2 min at 2.5 nM). Notably, the onset of intoxication at 37° became irreversible to washing within 2 min after exposure to BFT. Morphol. changes were completely inhibited by treatment of HT29/C1 cells with BFT at 4° but could be demonstrated by subsequent warming to temps. of 15° or higher after washing. The time required for the association of BFT with HT29/C1 cells at 4° but could be demonstrated by subsequent warming to temps. of 15° or higher after washing. The time required for the association of BFT with HT29/C1 4° was inversely correlated with concentration Inhibitors of endosomal and Golgi trafficking (NH4Cl and brefeldin A) prevented the intoxication of HT29/C1 cells by Clostridium difficile toxin A and cholera toxin, resp., but not by BFT. Agents altering microtubule structure did not affect the cellular activity of BFT. These data indicate that a purified toxin from B. fragilis strains associated with diarrhea rapidly and irreversibly intoxicates human intestinal epithelial cells (HT29/C1) in a concentration- and temperature-dependent manner and that the process of
- intoxication may not involve internalization mechanisms utilizing microtubules or
- sensitive to pH or brefeldin A.
 AN 1996:718615 HCAPLUS <<LOGINID::20080722>>
- DN 126:15745
- OREF 126:3225a,3228a
- TI Bacteroides fragilis toxin rapidly intoxicates human intestinal epithelial cells (HT29/C1) in vitro
- AU Saidi, Roxan F.; Sears, Cynthia L.
- CS Div. Gastroenterol. Infectious Diseases, Johns Hopkins Univ. Sch. Med., Baltimore, MD, 21205, USA
- SO Infection and Immunity (1996), 64(12), 5029-5034
- CODEN: INFIBR; ISSN: 0019-9567
- PB American Society for Microbiology
- DT Journal LA English
- RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L17 ANSWER 14 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Human intestinal epithelial cells swell and demonstrate actin

- rearrangement in response to the metalloprotease toxin of Bacteroides fragilis
- AB Enterotoxigenic Bacteroides fragilis (ETBF) cells produce a 20-kDa heat-labile metalloprotease toxin which is potentially important in the pathogenesis of diarrhea associated with this infection. Previous studies indicate that subconfluent HT29/C1 cells treated with the B. fragilis toxin (BFT) develop morphol. changes with dissoln. of tight clusters and apparent swelling. Such alterations suggest toxin-stimulated reorganization of the cellular cytoskeleton. The purpose of the current study was to evaluate the effect of B. fragilis toxin (BFT) on actin microfilaments (F-actin) and cell volume As assessed by fluorescent phallicidin staining which detects F-actin, BFT treatment of HT29/C1 cells resulted in redistribution of F-actin with loss of stress fibers, a floccular staining pattern, and cellular membrane blebbing without quant. changes in F-actin fluorescence intensity. The F-actin redistribution was time and concentration dependent. In contrast to the

cell shrinkage observed in response to the F-actin-depolymq. agents cytochalasin D and Clostridium difficile toxin A, BFT stimulated an increase in HT29/C1 cell volume of 10 to 25% (compared with control cells) over a 24-h time course. Only 10 to 30 ng of BFT per mL was necessary to stimulate a maximal increase in HT29/C1 cell volume The effect of BFT on cell volume was persistent and dependent on the proteolytic activity of BFT. In agreement with cell viability assays indicating that BFT did not injure HT29/C1 cells, intoxicated cells exhibited regulatory volume decrease, suggesting that toxin-treated cells remain physiol. dynamic. We conclude that BFT acts on the intestinal epithelial cell cytoskeleton to alter F-actin structure and to stimulate an increase in HT29/C1 cell volume Although these two activities of BFT appear to be linked, the precise sequence of cellular events following intoxication of HT29/C1 cells with BFT remains unclear. We hypothesize that these F-actin and cell volume changes may lead to an alteration in tight junction function in the polarized intestinal epithelium, contributing to the pathogenesis of diarrhea in ETBF infections.

AN 1996:718610 HCAPLUS <<LOGINID::20080722>>

DN 126:15744

OREF 126:3225a,3228a

I Human intestinal epithelial cells swell and demonstrate actin rearrangement in response to the metalloprotease toxin of Bacteroides fragilis

AU Koshy, Sherin S.; Montrose, Marshall H.; Sears, Cynthia L.

- CS Div. Infectious diseases Gastroenterol., Johns Hopkins Univ. Sch. Med., Baltimore, MD, 21205-2196, USA
- SO Infection and Immunity (1996), 64(12), 5022-5028 CODEN: INFIBR; ISSN: 0019-9567

PB American Society for Microbiology

DT Journal

LA English

RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

- L17 ANSWER 15 OF 15 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Human colon epithelial cells productively infected with human
- immunodeficiency virus show impaired differentiation and altered secretion

 Selected strains of the human immunodeficiency virus (HIV) types I and 2

 are able to infect human colon epithelial cells in vitro, suggesting a

 mechanism for the anal route of HIV transmission. In some cases, HIV is

 not produced by infected colon cells but can be rescued after coculture

 with T-lymphoid cells. One of the HIV strains (HIVI-NDK) replicated well

 in colonic cells. A transmission electron microscope study demonstrated

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two major structural perturbations in producer colon cells: an unusual number
     of secretion bodies and the appearance of intracellular lumina with
     disorganized microvilli, indicating a defect in brush border assembly and
     differentiation. Either abnormality could account for HIV-induced
     enteropathy consisting of chronic diarrhea and malabsorption in
     the absence of enteric pathogens. Moreover, HT29 cells infected
     with HIV provide a unique model for selection of enterotropic HIV strains.
AΝ
    1992:57208 HCAPLUS <<LOGINID::20080722>>
DN
     116:57208
OREF 116:9895a,9898a
     Human colon epithelial cells productively infected with human
     immunodeficiency virus show impaired differentiation and altered secretion
     Fantini, Jacques; Yahi, Nouara; Baghdiquian, Stephen; Chermann, Jean
     Claude
     Univ. Aix-Marseille I, Marseille, 13331, Fr.
SO.
    Journal of Virology (1992), 66(1), 580-5
     CODEN: JOVIAM; ISSN: 0022-538X
    Journal
LA
    English
=> d his
     (FILE 'HOME' ENTERED AT 13:29:10 ON 22 JUL 2008)
     FILE 'HCAPLUS' ENTERED AT 13:30:24 ON 22 JUL 2008
           3287 S URSODEOXYCHOL?
         139310 S ISCHEM? OR STROKE OR NEUROPROTECTIVE
             28 S L1 AND L2
             19 S L3 AND (PY<2005 OR AY<2005 OR PRY<2005)
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     FILE 'STNGUIDE' ENTERED AT 13:30:46 ON 22 JUL 2008
     FILE 'HCAPLUS' ENTERED AT 15:30:51 ON 22 JUL 2008
L5
         96359 S PREBIOTIC OR ENTERAL OR DIARRHEA OR NUTRITIONAL
         493913 S ADHESIVE OR ADHESION
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     FILE 'HCAPLUS' ENTERED AT 15:31:22 ON 22 JUL 2008
          95742 S ((ARABINO OR MANNO OR GALACTO OR ISOMALTO OR SIALYL)(W)OLIGOS
             19 S L5 AND L6 AND L7
     FILE 'STNGUIDE' ENTERED AT 15:31:26 ON 22 JUL 2008
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              8 S L8 AND (PY<2004 OR AY<2004 OR PRY<2004)
     FILE 'STNGUIDE' ENTERED AT 15:32:20 ON 22 JUL 2008
     FILE 'HCAPLUS' ENTERED AT 15:32:30 ON 22 JUL 2008
     FILE 'STNGUIDE' ENTERED AT 15:32:31 ON 22 JUL 2008
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T.10
          2473 S HT29
             1 S L5 AND L6 AND L10
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ΑU

DТ

L2

L3

L4

1.6

1.8

L9

L12 1 S L5 AND L7 AND L10 L13 0 S L11 AND (PY<2004 OR AY<2004 OR PRY<2004) L14 0 S L12 AND (PY<2004 OR AY<2004 OR PRY<2004) FILE 'STNGUIDE' ENTERED AT 15:51:16 ON 22 JUL 2008 FILE 'HCAPLUS' ENTERED AT 15:51:30 ON 22 JUL 2008 FILE 'STNGUIDE' ENTERED AT 15:51:30 ON 22 JUL 2008 FILE 'HCAPLUS' ENTERED AT 15:51:33 ON 22 JUL 2008 FILE 'STNGUIDE' ENTERED AT 15:51:33 ON 22 JUL 2008 FILE 'HCAPLUS' ENTERED AT 15:51:41 ON 22 JUL 2008 FILE 'STNGUIDE' ENTERED AT 15:51:41 ON 22 JUL 2008 FILE 'HCAPLUS' ENTERED AT 15:53:20 ON 22 JUL 2008 L15 26 S L5 AND L10 L16 188 S L6 AND L10 L17 15 S L15 AND (PY<2004 OR AY<2004 OR PRY<2004) L18 115 S L16 AND (PY<2004 OR AY<2004 OR PRY<2004) FILE 'STNGUIDE' ENTERED AT 15:53:29 ON 22 JUL 2008 FILE 'HCAPLUS' ENTERED AT 15:53:39 ON 22 JUL 2008 FILE 'STNGUIDE' ENTERED AT 15:53:40 ON 22 JUL 2008

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FULL ESTIMATED COST	0.06	162.36
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
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=> exp mannobiose/cn
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E1	1	MANNOBIITOL/CN
E2	1	MANNOBIOHYDROLASE, EXO-1,4-B-/CN
E3	1>	MANNOBIOSE/CN
E4	1	MANNOBIOSE, 1,6-A-/CN
E5	1	MANNODEXTRIN/CN
E6	1	MANNOFRUCTOKINASE/CN
E7	1	MANNOFUCOGALACTAN/CN
E8	1	MANNOFURANOSE, 1,1'-DITHIOBIS(1-DEOXY-2,3:5,6-DI-O-ISOPROPYL
		IDENE-, A-D-/CN
E9	1	MANNOFURANOSE, 1,2:5,6-DI-O-ISOPROPYLIDENE-, B-D-/CN
E10	1	MANNOFURANOSE, 1,2:5,6-DI-O-ISOPROPYLIDENE-, ACETATE, B
		-D-/CN
E11	1	MANNOFURANOSE, 1,5,6-TRIACETATE CYCLIC 2,3-CARBONATE/CN
E12	1	MANNOFURANOSE, 1,5,6-TRIACETATE CYCLIC 2,3-CARBONATE, .ALPHA
	_	.=D-/CN
		/

=> s E4

1 "MANNOBIOSE, 1,6-A-"/CN

=> exp alpha mannobiose/cn

E1 4 ALPHA KG DEPENDENT 2,4-D DIOXYGENASE (BURKHOLDERIA XENOVORAN S STRAIN LB400)/CN

```
E2
                   ALPHA LIPID 300/CN
E3
             0 --> ALPHA MANNOBIOSE/CN
E4
                   ALPHA MANNOSIDASE (SYNECHOCOCCUS STRAIN WH8102 GENE SYNW0267
             1
                   ALPHA MANNOSIDASE 6A8B (HUMAN GENE 6A8B)/CN
E5
                   ALPHA MANNOSIDASE II ISOZYME (HUMAN CELL LINE SK-MEL-28 CLON
E6
             1
                   E PMX6)/CN
E7
                   ALPHA MANNOSIDASE II ISOZYME (HUMAN CELL LINE SK-MEL-28)/CN
E8
                   ALPHA MATING PHEROMONE (SACCHAROMYCES NAGANISHII GENE MFALPH
                  A1 PRECURSOR)/CN
E9
                  ALPHA MEDOPA/CN
E10
             1
                   ALPHA METALS 171/CN
E11
                   ALPHA MS/CN
             1
E12
                  ALPHA NAC (ARABIDOPSIS THALIANA GENE F7L13.60)/CN
             1
=> exp alpha 1,3-mannobiose/cn
E1
                   ALPHA 1,3-FUCOSYLTRANSFERASE (HELICOBACTER PYLORI STRAIN HPA
                   G1)/CN
                   ALPHA 1,3-FUCOSYLTRANSFERASE FUC-T (SIMILAR TO MOUSE FUT4) (
                   RATTUS NORVEGICUS CLONE MGC:72456 IMAGE:5621698)/CN
             0 --> ALPHA 1,3-MANNOBIOSE/CN
E4
             1
                   ALPHA 1,4-GALACTOSYLTRANSFERASE (HUMAN CLONE MGC:9631 IMAGE:
                   3913851)/CN
E5
             1
                   ALPHA 1-6-GLUCOSIDASE (EC 3.2.1.70) (LACTOCOCCUS LACTIS LACT
                   IS STRAIN IL1403 GENE DEXB)/CN
E6
                   ALPHA 1-ANTITRYPSIN (HUMAN)/CN
                   ALPHA 100/CN
             1
E8
             1
                  ALPHA 1000/CN
E9
             1
                  ALPHA 127/CN
E10
                  ALPHA 1800/CN
             1
E11
             1
                  ALPHA 1C ADRENERGIC RECEPTOR ISOFORM 2 (HUMAN CLONE P2C6)/CN
                  ALPHA 2 ACTIN (HUMAN CLONE MGC:9221 IMAGE:3906861)/CN
             1
=> exp alpha 1,3 mannobiose/cn
             1
                   ALPHA 1,2-MANNOSIDASE (HUMAN CLONE MGC:12553 IMAGE:3959196)/
E2
             1
                   ALPHA 1,2-MANNOSIDASE IB (HUMAN)/CN
E3
             0 --> ALPHA 1,3 MANNOBIOSE/CN
E4
                   ALPHA 1,3-FUCOSYLTRANSFERASE (HELICOBACTER PYLORI STRAIN HPA
                   G1)/CN
             1
                   ALPHA 1,3-FUCOSYLTRANSFERASE FUC-T (SIMILAR TO MOUSE FUT4) (
                   RATTUS NORVEGICUS CLONE MGC:72456 IMAGE:5621698)/CN
E6
             1
                   ALPHA 1,4-GALACTOSYLTRANSFERASE (HUMAN CLONE MGC:9631 IMAGE:
                   3913851)/CN
E7
             1
                   ALPHA 1-6-GLUCOSIDASE (EC 3.2.1.70) (LACTOCOCCUS LACTIS LACT
                   IS STRAIN IL1403 GENE DEXB)/CN
                  ALPHA 1-ANTITRYPSIN (HUMAN)/CN
E8
             1
                  ALPHA 100/CN
E9
                   ALPHA 1000/CN
E10
E11
                   ALPHA 127/CN
                   ALPHA 1800/CN
             1
=> exp 1,3 mannobiose/cn
E1
                   1.2R, 3.4S, 5-PENTAAMMONIOPENTANE TETRACHLOROZINCATE TRICHLORI
                   DE MONOHYDRATE/CN
E 2
                   1,3 BENZENEDICARBOXYLIC ACID, POLYMER WITH 2-ETHYL-2-(HYDROX
                   YMETHYL)-1,3-PROPANEDIOL, HEXANEDIOIC ACID, 1,6-HEXANEDIOL,
                   1,3-ISOBENZOFURANDIONE AND 1,1'-METHYLENEBIS(4-ISOCYANATOBEN
                   ZENE), DI-ET MALONAT/CN
             0 --> 1.3 MANNOBIOSE/CN
E3
E.4
                  1.3 PROPANEDIOL DEHYDROGENASE (GEOBACILLUS KAUSTOPHILUS STRA
```

```
IN HTA426)/CN
E5
                  1,3'(2H,2'H)-SPIROBI(CYCLOPENT(B)INDOLE)/CN
E6
                   1,3'(2H,2'H)-SPIROBI(CYCLOPENT(B)INDOLE), 1',3,4,4'-TETRAHYD
                  RO-1',1',3,3-TETRAMETHYL-4,4'-BIS(PHENYLMETHYL)-/CN
                  1.3'.3'-TRIMETHYL-6-NITRO-8-BROMOSPIRO(2H-1-BENZOPYRAN-2,2'-
                  INDOLINE) / CN
                  1,3'-(BIPYRROLIDIN)-5'-ONE, 4',4'-DIMETHYL-2'-((3-NITRO-O-TO
E8
                  LYL) IMINO) - 1 '-PHENYL-/CN
E9
                  1,3'-BI-1,2,4-TRIAZOLE, 3,5-DIMETHYL-/CN
E10
                  1,3'-BI-1,2-DICARBADODECABORANE(12)/CN
E11
                  1,3'-BI-1,2-DICARBADODECABORANE(12), 1'-METHYL-/CN
E12
             1
                  1,3'-BI-1,2-DICARBADODECABORANE(12), 2-METHYL-/CN
=> exp alpha 1,2-mannobiose/cn
E1
                  ALPHA 1,2 N-ACETYLGLUCOSAMINE TRANSFERASE (NEISSERIA MENINGI
                  TIDIS GROUP C STRAIN FAM18 GENE RFAK)/CN
                   ALPHA 1,2 N-ACETYLGLUCOSAMINE TRANSFERASE (SYMBIOBACTERIUM T
E 2
                  HERMOPHILUM STRAIN IAM14863)/CN
E3
             0 --> ALPHA 1,2-MANNOBIOSE/CN
Ε4
                  ALPHA 1,2-MANNOSIDASE (HUMAN CLONE MGC:1215 IMAGE:3533651)/C
E5
             1
                  ALPHA 1,2-MANNOSIDASE (HUMAN CLONE MGC:12553 IMAGE:3959196)/
                  CN
                  ALPHA 1,2-MANNOSIDASE IB (HUMAN)/CN
                  ALPHA 1.3-FUCOSYLTRANSFERASE (HELICOBACTER PYLORI STRAIN HPA
E7
                  G1)/CN
                  ALPHA 1,3-FUCOSYLTRANSFERASE FUC-T (SIMILAR TO MOUSE FUT4) (
E8
                  RATTUS NORVEGICUS CLONE MGC:72456 IMAGE:5621698)/CN
E9
             1
                  ALPHA 1,4-GALACTOSYLTRANSFERASE (HUMAN CLONE MGC:9631 IMAGE:
                  3913851)/CN
E10
             1
                  ALPHA 1-6-GLUCOSIDASE (EC 3.2.1.70) (LACTOCOCCUS LACTIS LACT
                  IS STRAIN IL1403 GENE DEXB)/CN
                  ALPHA 1-ANTITRYPSIN (HUMAN)/CN
             1
E12
                  ALPHA 100/CN
             1
=> exp alpha 1,6-mannobiose/cn
             1
                  ALPHA 1,3-FUCOSYLTRANSFERASE FUC-T (SIMILAR TO MOUSE FUT4) (
                  RATTUS NORVEGICUS CLONE MGC:72456 IMAGE:5621698)/CN
                  ALPHA 1,4-GALACTOSYLTRANSFERASE (HUMAN CLONE MGC:9631 IMAGE:
             1
                  3913851)/CN
E3
             0 --> ALPHA 1,6-MANNOBIOSE/CN
E4
             1
                  ALPHA 1-6-GLUCOSIDASE (EC 3.2.1.70) (LACTOCOCCUS LACTIS LACT
                  IS STRAIN IL1403 GENE DEXB)/CN
E5
                 ALPHA 1-ANTITRYPSIN (HUMAN)/CN
E6
                 ALPHA 100/CN
E7
                 ALPHA 1000/CN
            1
E8
                 ALPHA 127/CN
            1
                 ALPHA 1800/CN
E9
            1
                 ALPHA 1C ADRENERGIC RECEPTOR ISOFORM 2 (HUMAN CLONE P2C6)/CN
E10
            1
E11
                 ALPHA 2 ACTIN (HUMAN CLONE MGC:9221 IMAGE:3906861)/CN
                  ALPHA 2 BI-ACTIVE-ENERGIE 5/CN
            1
=> exp mannooligosacch/cn
E1
                  MANNONOYL CHLORIDE, PENTAACETATE/CN
E2
                  MANNONOYL CHLORIDE, PENTAACETATE, L-/CN
E3
             0 --> MANNOOLIGOSACCH/CN
E4
            1
                 MANNOPENTAOSE/CN
E5
            1
                 MANNOPENTAOSE SULFATE/CN
                 MANNOPENTAOSE-DI (N-ACETYL-D-GLUCOSAMINE) /CN
E6
            1
E7
            1
                 MANNOPEPTIMYCIN A/CN
                 MANNOPEPTIMYCIN B/CN
ER
            1
```

E9	1	MANNOPEPTIMYCIN A/CN
E10	1	MANNOPEPTIMYCIN E/CN
E11	1	MANNOPEPTIMYCIN F/CN
E12	1	MANNODEDTIN A /CN

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=> s 119/cn

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=> s 119/thu

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1030340 THU/RL
0 L19/THU
(L19 (L) THU/RL)
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L21 1 L19 => d 121 ti abs bib

T.20

=> s 119

- L21 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2008 ACS on STN
- TI The configuration of glycosidic linkages in oligosaccharides. I. Application of Jackson and Hudson's oxidation method to reducing disacreparides.
- disaccharides AB Disaccharides were degraded by glycol-cleavage oxidations, on the premise that all disaccharides of a given class would yield the same product. The relative contribution of the glycosidic center to the optical activity is enhanced in the oxidation products, and thereby promotes a larger rotational difference between α - and β - compds. than in the original disaccharides. All α - and β -Me aldohexopyranosides of the D-series yield OHCCH(CH2OH)OCH(OMe)CHO (I) by cleavage of the 2,3,4-triol group; α - and β -glycosides give aldehydes with large pos. and neg. specific rotation, resp., which differ only in configuration at the glycosidic center. D-Aldohexopyranose disaccharides having 1,6-linkages are degraded by IO4- (II) or Pb(OAc)4 (III) to OHCCH(CH2OH)OCH(CHO)OCH2CHO (IV) in which the reducing end-unit has been converted to HOCH2CHO and the glycosidic residue yielded a dialdehyde similar to I. A 1,4-hexose disaccharide such as cellobiose (V) should be converted by III to a structure (VI) in which the D-erythrose (VII) unit of the reducing end is linked at the 2-position to a dialdehyde similar to I, and related in configuration to I derived from $\beta-D$ aldohexopyranosides. Maltose (VIII) should yield a compound which differs from VI only in the configuration of the glycosidic center. All D-aldohexopyranose disaccharides in which the reducing end is D-glycose (IX) or D-mannose (X) should yield one of these two oxidation products depending on the configuration of the biose linkage. If D-galactose (XI) is the reducing end-unit, the cleavage product should be a compound similar to VI, in which VII is replaced by D-threose. The results obtained by glycol-cleavage oxidation were (disaccharide, [α]D disaccharide, oxidizing agent, and $[\alpha]D$ oxidized disaccharide given): melibiose (XII), 129°, II, 79°; isomaltose (XIII), 98°, II, 85°; mannobiose (1,6α) 50°, II, 88°; galactosido-erythritol (1,4a), 134°, II, 79°; gentiobiose (XIV), 8°, II, -109°; mannosido-erythrito] (1,4β), -39°, II, -106°; VIII, 130°, III, 24°; glucosido-arabinose (1,3a), 48°, III, 19°; lactose (XV), 55°, III, -78° V, 34°, III, -80°; glucosido-mannose (1,4B), 47°, III, -71°; mannobiose (1,4β), -6°, III, -84°; glucosido-erythritol $(1,2\alpha)$, 130° II, 5°; glucosido-erythritol $(1,2\beta)$, -17° II, 0; galactosido-erythritol (1,2β), 7° II, 0; glucosido-fructose (1,5a), -8°, II, 36°; xylobiose (1,4β), -25°, II, 102°. Disaccharides having 1,2-linkages are over-oxidized by II and III and could not be examined by direct oxidation. Three such compds. were reduced to the alc., in order to avoid over-oxidation, and then treated with II. The products gave zero or very small rotations (see above). This was attributed to the formation of symmetrical products such as OHCCH[OCH(CHO)CH2OH]2. It is concluded that the method is not applicable to 1,2- or 1,5-hexopyranose disaccharides. XIII and XIV were prepared from the crystalline octaacetates by deacetylation with NaOMe. The sirups were neutralized with HOAc (XVI), the MeOH evaporated, and the sirups used directly in the oxidations. In a

```
typical experiment, 136 mg. XII hydrate in 5 ml. H2O was mixed with 600 mg.
     NaIO4 in 5 ml. H2O; αD was almost constant for 4 hrs. at 1.26°,
     but dropped to 1.13° after 24 hrs. Assuming conversion of XII to
     72 mg. IV, IV gave [\alpha]D 79°. Oxidations with III were
     carried out at 28° in a constant volume Warburg apparatus. In a typical
     experiment, 9 manometers were used; each flask contained 1.5 mg. VIII in 0.2
     ml. 90% XVI and 25 mg. III and 10 mg. KOAc (XVII) in 1.0 ml. 90% XVI. The
     CO2 evolved indicated the formation of 1 mole HCO2H in 5-6 hrs. The
     contents of all the flasks were combined, excess III destroyed by addition of
     0.5 ml. (CO2H)2 (XVIII) (10% in XVI), Pb(C2O4)2 (XIX) filtered off and
     washed with XVI, and the filtrate concentrated to dryness at 40°. H2O (3
     ml.) was added to the residue and the slightly turbid solution was stored for
     18 hrs. at 3° to promote hydrolysis of the HCO2R groups, following
     which the solution was neutralized with Pb(CO3)2 (XX) and filtered through
     Celite. The clear filtrate was used for polarimetric measurements (2-dm.
     tube); aD 0.17° (initial) to 0.15° (24 hrs.; constant);
     this corresponded to [a]D27 23° assuming conversion of VIII
     to 9.9 mg. of the α-anomer of VI. Expts. with Me glycosides
     indicated that the loss of oxidation product using III was small. Me
     β-D-glucopyranoside gave values of [α]D27 -123° and
     -132° compared with values of -150° and -141° by
     oxidation with II. Larger-scale oxidations with III were carried out in
     glass-stoppered Erlenmever flasks. In a typical experiment, 51.1 mg. XV
     hydrate in 10 ml. 90% XVI was mixed with 750 mg. III and 300 mg. XVI in 30
     ml. 90% XVI. After 3 hrs., a 2.0 ml. aliquot showed that 4.9 moles III
     had reacted (theory 5.0 moles). Optical rotation was determined on an aqueous
     solution prepared as described above; αD -0.96° (initial) to
     -1.03° (24 hrs.; constant). [a]D of VI was -76° assuming
     conversion of 48.6 mg. XV hydrate to 33.8 mg. VI. V, VIII, and XV hydrate
     were oxidized with III as described above, meso-(CHMeOH)2 being used in
     place of XVIII, and with the omission of the use of XX. The hydrolysis
     rate of the formate esters of each product was measured polarimetrically.
     The results were (initial concentration of sugar, 1% in a 2-dm. tube at
     27°) (αD at 0.5, 5, 24, 48, and 72 hrs., and [α]D
     given): VIII, 1.05°, 0.86°, 0.54° 0.47°,
     0.46°, 31°; V, 0.05°, -0.38°, -0.98°,
     -1.15° -1.13°, -77° XV, 0.10°, -0.34°,
     -0.86°, -1.06°, -1.06, -73°.
    1957:43153 CAPLUS <<LOGINID::20080722>>
    51:43153
OREF 51:8016c-i,8017a-e
    The configuration of glycosidic linkages in oligosaccharides. I.
    Application of Jackson and Hudson's oxidation method to reducing
     disaccharides
    Charlson, A. J.; Perlin, A. S.
    Natl. Research Council, Saskatoon, SK
    Canadian Journal of Chemistry (1956), 34, 1804-10
     CODEN: CJCHAG; ISSN: 0008-4042
     Journal
    Unavailable
=> s mannobiose
L22
          380 MANNOBIOSE
=> file registry
COST IN U.S. DOLLARS
                                                SINCE FILE
                                                                 TOTAL
                                                      ENTRY
                                                              SESSION
FULL ESTIMATED COST
                                                       8.59
                                                               178.42
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)
                                               SINCE FILE
                                                                TOTAL
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=> s mannobiose/cn

L23 1 MANNOBIOSE/CN

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=> d 124 1-6 ti abs bib

- L24 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
- TI Anti-inflammatory compositions or agents containing $\beta\text{--}1,4\text{--mannobiose}$ and food or feed containing them
- AB Title compns. or agents are useful for prevention or amelioration of sepsis, inflammatory diseases such as rheumatoid arthritis, inflammatory bowel disease, etc., through suppression of IL-8 production Thus, copra meal was treated with Hemicellulase GM Amano at 60° for 12 h and dried to water content 10%. The powder thus obtained was extracted with EtOH 3 times, the residue was extracted with warm water at 60°, filtered, and the filtrate was freeze-dried to give a composition containing 21.74% B-1,4-mannobiose. The composition significantly suppressed LPS-induced IL-8 production by Caco-2 cells.
- AN 2008:63936 CAPLUS <<LOGINID::20080722>>
- DN 148:143661
- TI Anti-inflammatory compositions or agents containing β -1,4-mannobiose and food or feed containing them
- IN Yokomizo, Futoshi; Ibuki, Masahisa; Mine, Yoshinori; Katayama, Shigeru
- PA Fuji Oil Co., Ltd., Japan
- SO Jpn. Kokai Tokkyo Koho, 7pp.
- CODEN: JKXXAF
- DT Patent
- LA Japanese
- FAN CNT 1

PAN.CNI I				
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2008007505	A	20080117	JP 2007-167720	20070626
PRAI US 2006-80575	IP P	20060626		

- L24 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
- TI Antiallergic composition and agent, and food, beverage and animal feed each containing the composition or agent
- AB The invention provides an anti-allergic composition or agent comprising β -1,4-mannobiose. The composition containing β -1,4-mannobiose is prepared by degradation of mannan containing natural products derived from coconut cake, copra meal and palm kernel meal with mannan-degrading enzyme hemicellulase. The composition containing β -1,4-mannobiose is then dried and extracted with ethanol. The composition containing β -1,4-mannobiose has an inhibitory effect on mast cell degranulation. The composition containing β -1,4-mannobiose is added to foods, beverages and animal feeds.
- AN 2008:11239 CAPLUS <<LOGINID::20080722>>
- DN 148:77751
 - Antiallergic composition and agent, and food, beverage and animal feed each containing the composition or agent
- IN Yokomizo, Futoshi; Ibuki, Masahisa; Mine, Yoshinori; Katayama, Shigeru
- PA Fuji Oil Company, Limited, Japan
- SO PCT Int. Appl., 18pp.
- CODEN: PIXXD2

DT Patent LA Japanese FAN.CNT 1

	PA:	PATENT NO.					KIND DATE			APPLICATION NO.						DATE			
PI	WO	2008	0017	70		A1		2008	0103	,	WO 2	007-	JP62:	800		20070626			
		W:	ΑE,	AG,	AL,	AM,	ΑT,	AU, AZ,		BA,	BB,	BG,	BH,	BR,	BW,	BY,	BZ,	CA,	
			CH,	CN,	CO,	CR,	CU,	CZ,	DE,	DK,	DM,	DO,	DZ,	EC,	EE,	EG,	ES,	FΙ,	
			GB,	GD,	GE,	GH,	GM,	GT,	HN,	HR,	HU,	ID,	IL,	IN,	IS,	JP,	KE,	KG,	
			KM,	KN,	KP,	KR,	ΚZ,	LA,	LC,	LK,	LR,	LS,	LT,	LU,	LY,	MA,	MD,	ME,	
			MG,	MK,	MN,	MW,	MX,	MY,	ΜZ,	NA,	NG,	NI,	NO,	NZ,	OM,	PG,	PH,	PL,	
			PT,	RO,	RS,	RU,	SC,	SD,	SE,	SG,	SK,	SL,	SM,	SV,	SY,	TJ,	TM,	TN,	
			TR,	TT,	TZ,	UA,	UG,	US,	UΖ,	VC,	VN,	ZA,	ZM,	zw					
		RW:	ΑT,	BE,	BG,	CH,	CY,	CZ,	DE,	DK,	EE,	ES,	FI,	FR,	GB,	GR,	HU,	ΙE,	
			IS,	IT,	LT,	LU,	LV,	MC,	MT,	NL,	PL,	PT,	RO,	SE,	SI,	SK,	TR,	BF,	
			ΒJ,	CF,	CG,	CI,	CM,	GA,	GN,	GQ,	GW,	ML,	MR,	ΝE,	SN,	TD,	TG,	BW,	
			GH,	GM,	KE,	LS,	MW,	ΜZ,	NA,	SD,	SL,	SZ,	TZ,	UG,	ZM,	ZW,	AM,	ΑZ,	
			BY,	KG,	ΚZ,	MD,	RU,	TJ,	TM										
PRAI	I US 2006-805753P P 20060626																		

RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L24 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN

TΙ Intestinal immunity-activating substance or agent, and food, beverage and animal feed containing the same

AB The invention provides an intestinal immunity-activating substance or agent containing \$\textit{\beta}\$-1,4-mannobiose. The intestinal immunity-activating substance containing β -1,4-mannobiose is prepared by degradation of mannan containing natural products derived from coconut cake, copra meal or palm kernel meal with mannan-degrading enzyme hemicellulase. The intestinal immunity-activating substance containing $\beta-1$, 4-mannobiose is then dried and extracted with ethanol. The substance containing $\beta-1$, 4-mannobiose enhances IgA production, has a preventive effect on diseases caused by pathogenic bacteria and viruses and also has a preventive effect on allergy. The substance containing $\beta-1$, 4-mannobiose is added to foods, beverages and animal feeds.

2008:10173 CAPLUS <<LOGINID::20080722>> AN

DN 148:77749

TΙ Intestinal immunity-activating substance or agent, and food, beverage and animal feed containing the same

IN Yokomizo, Futoshi; Ibuki, Masahisa; Mine, Yoshinori; Katayama, Shigeru Fuji Oil Company, Limited, Japan PA

SO PCT Int. Appl., 17pp.

CODEN: PIXXD2

DT Patent

LA Japanese

FAN.	CNT	1																			
	PA:	ENT :	NO.			KIND DATE			- 2	APPL	ICAT	ION	NO.		DATE						
PI	WO	2008	0017	69		A1		20080103		1	WO 2007-JP62799					20070626					
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			GH,	GM,	KE,	LS,	MW,	MZ,	NA,	SD,	SL,	SZ,	TZ,	UG,	ZM,	ZW,	AM,	AZ,			

BY, KG, KZ, MD, RU, TJ, TM

PRAI US 2006-805752P P 20060626

RE.CNT 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

- L24 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
- Mechanism of glycofection: enhanced nuclear import of plasmid DNA complex with disaccharide-conjugated poly(ethylenimine)s
- AB The mechanism of nuclear import of glycoconjugates consisting of disaccharide-modified polyethylenimine (PEI)/DNA polyplexes was studied using cytoplasmic microinjection. Eight reductive disaccharides (lactose, maltose, isomaltose, mannobiose, melibiose, gentiobiose, cellobiose, and laminaribiose) were conjugated with branched PEI by reductive amination. Cytoplasmic microinjection of fluorescent labeled polyplex showed that 28Lac-42Lac-PEI, and 9.3Cel-PEI showed no effect for the nuclear import of plasmid DNA. Lactose-conjugation to cationic non-viral vector increased the transfection efficiency by the enhancement to internalization of polyplex and nuclear import of gene. Nuclear import of glucopolyplex was distinct from importin-β-dependent mechanism.
- AN 2006:887556 CAPLUS <<LOGINID::20080722>>
- 146:407763 DN
- TI Mechanism of glycofection: enhanced nuclear import of plasmid DNA complex with disaccharide-conjugated poly(ethylenimine)s
- Nagasaki, Takeshi; Shiga, Toshiki; Jinta, Tomomi; Shinkal, Seiji
- CS Department of Bioengineering, Graduate School of Engineering, Osaka City University, Osaka, Japan
- PMSE Preprints (2006), 95, 667 SO
- CODEN: PPMRA9; ISSN: 1550-6703
- PB American Chemical Society
- DT Journal; (computer optical disk)
- LA English
- L24 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
- Body fat-reducing compositions containing mannooligosaccharides, and foods and beverages containing them
- AR Title compns. contain mannose-based oligosaccharides with d.p. 1-10.
- Thus, coffee containing 1 g/100 mL mannooligosaccharide composition (prepared by high-pressure steam treatment of extraction residue of ground coffee beans)
- significantly reduced the size of s.c. fat area in volunteers. AN 2006:294389 CAPLUS <<LOGINID::20080722>>
- DN 144:330463
- ΤI Body fat-reducing compositions containing mannooligosaccharides, and foods and beverages containing them
- IN Asano, Ichiro; Fujii, Shiqeyoshi; Muto, Katsuhito; Takao, Izumi; Ozaki, Kazuto; Nakamuro, Kenichi; Matsushima, Toshiyuki
- Ajinomoto General Foods, Inc., Japan PA
- Jpn. Kokai Tokkvo Koho, 11 pp. CODEN: JKXXAF
- DT Patent.
- LA Japanese
- FAN.CNT 1

SO

	PATENT		KIND DATE				APPLICATION NO.						DATE					
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PI	JP 200€	0831	27		A		2006	0330		JP 2	004-	2714	12		2	0040	917	
	AU 2005	AU 2005290262			A1		20060406			AU 2005-290262					2005041			
	CA 2580652				A1		2006	0406		CA 2	005-	2580	652		2	20050414		
	WO 200€	0362	80		A1		2006	0406		WO 2	005-	US12	823		2	0050	414	
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                             20070725 EP 2005-734832
                       A1
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    CN 101087621
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    IN 2007CN01566
                       A
                             20070831
                                        IN 2007-CN1566
                                                              20070417
PRAI JP 2004-271412
                       A
                             20040917
    WO 2005-US12823
                       147
                             20050414
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- L24 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2008 ACS on STN
- TI Stabilized compositions of tissue-type plasminogen activator
- AB This invention relates to a composition containing natural or modified tissue-type

plasminogen activator (t-PA) and mannose derivs., for the improvement of stability, soly, and half-life of t-PA. A solution was formulated containing t-PA 0.5 mg/mL, NaH2PO4/Na2HPO4 0.1 M (pH 7.2), NaCl 0.8 %, β -1,4-mannobiose 10 %, and Tween 80 0.01 % and freeze-dried.

- AN 2001:217889 CAPLUS <<LOGINID::20080722>>
- DN 134:242690
- TI Stabilized compositions of tissue-type plasminogen activator
- IN Tanaka, Junya; Yoshikawa, Genichi; Mukai, Katsuyuki
- PA Unitika Ltd., Japan
- SO Jpn. Kokai Tokkyo Koho, 6 pp.
- CODEN: JKXXAF
- DT Patent LA Japanese
- FAN.CNT 1

PATE	NT NO.	KIND	DATE	ΑE	PPLICATION NO.	DATE			
PI JP 2	001081040	A	20010327	JE	1999-256201	19990909			
PRAT JP 1	999-256201		19990909						

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 COST IN U.S. DOLLARS
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 FULL ESTIMATED COST
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FULL ESTIMATED COST 0.12 204.21

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CA SUBSCRIBER PRICE 0.00 -40.80

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=> s methyl alpha manno? 1060565 METHYL 1779675 ALPHA 58520 MANNO?

L25 64 METHYL ALPHA MANNO?

(METHYL (W) ALPHA (W) MANNO?)

=> s nutritional or enteral or prebiotic

66844 NUTRITIONAL 4337 ENTERAL

4374 PREBIOTIC

L26 74681 NUTRITIONAL OR ENTERAL OR PREBIOTIC

=> s 125 and 126 L27 0 L25 A

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CA SUBSCRIBER PRICE 0.00 -40.80

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This file contains CAS Registry Numbers for easy and accurate substance identification.

=> s gut or intestine or oral or pharmaceutical

31419 GUT 217128 INTESTINE

228570 ORAL

300822 PHARMACEUTICAL

1.28 714245 GUT OR INTESTINE OR ORAL OR PHARMACEUTICAL

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COST IN U.S. DOLLARS SINCE FILE TOTAL ENTRY SESSION FULL ESTIMATED COST 2.69 209.65 SINCE FILE TOTAL ENTRY SESSION DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) CA SUBSCRIBER PRICE 0.00 -40.80 FILE 'STNGUIDE' ENTERED AT 16:46:43 ON 22 JUL 2008 USE IS SUBJECT TO THE TERMS OF YOUR CUSTOMER AGREEMENT COPYRIGHT (C) 2008 AMERICAN CHEMICAL SOCIETY (ACS)

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jul 18, 2008 (20080718/UP).

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YOU HAVE REQUESTED DATA FROM FILE 'HCAPLUS' - CONTINUE? (Y)/N:v

- L29 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2008 ACS on STN
- ${\tt TI} \quad {\tt Glycoconjugate} \ {\tt histochemistry} \ {\tt of} \ {\tt the} \ {\tt digestive} \ {\tt tract} \ {\tt of} \ {\tt Triturus} \ {\tt carnifex} \ ({\tt Amphibia, Caudata})$
- AB In this study, the varieties of sugar residues in the gut glycoconjugates of Triturus carnifex are investigated by carbohydrate conventional histochem. and lectin histochem. The esophageal surface mucous cells contained acidic glycoconjugates, with residues of GalNAc, Gal B1.3 GalNAc and (GlcNAc B1.4)n oligomers. The gastric surface cells mainly produced neutral glycoproteins with residues of fucose, Gal β1-3 GalNAc, Gal-αGal, and (GlcNAc β1,4)noligomers in N- and O-linked glycans, as the glandular mucous neck cells, with residues of mannose/glucose, GalNAc, Gal β1,3 GalNAc, (GlcNAc β1,4)n oligomers and fucose linked α1,6 or terminal al,3 or al,4 in O-linked glycans. The oxynticopeptic tubulo-vesicular system contained neutral glycoproteins with N- and O-linked glycans with residues of Gal-αGal, Gal β1-3 GalNAc and (GlcNAc β1,4)n oligomers; Fuc linked α1,2 to Gal, α1,3 to GlcNAc in (poly) lactosamine chains and 01,6 to GlcNAc in N-linked glycans. Most of these glycoproteins pribably corresponds to the H+K+-ATPase β-subunit. The intestinal goblet cells contained acidic glycoconjugates, with residues of GalNAc, mannose/ glucose, (GlcNAc β1,4)n oligomers and fucose linked α1,2 to Gal in O-linked oligosaccharides. The different composition of the mucus in the digestive tracts may be correlated with its different functions. In fact the presence of abundant sulfation of glycoconjugates, mainly in the esophagus and intestine, probably confers resistance to bacterial enzymic degradation of the mucus barrier.
- AN 2007:560386 HCAPLUS <<LOGINID::20080722>>
- DN 147:254134
- TI Glycoconjugate histochemistry of the digestive tract of Triturus carnifex (Amphibia, Caudata)
- AU Liquori, Giuseppa Esterina; Mastrodonato, Maria; Zizza, Sara; Ferri, Domenico
- CS Dipartimento di Zoologia, Laboratorio di Istologia e Anatomia comparata, Universita degli Studi di Bari, Bari, 70125, Italy
- SO Journal of Molecular Histology (2007), 38(3), 191-199 CODEN: JMHOAO; ISSN: 1567-2379
- PB Springer
- DT Journal
- LA English
- RE.CNT 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD
 ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L29 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Sequences of human C-type lectin DC-SIGN and DC-SIGNR, and uses thereof for inhibiting hepatitis C virus infection
- AB This invention provides protein sequences of human C-type lectin DC-SIGN,

DC-SIGNR, and hepatitis C virus polyprotein. This invention further provides a method of inhibiting HCV infection of a cell susceptible to HCV infection which comprises contacting the cell with an amount of a compound effective to inhibit binding of an HCV envelope glycoprotein to a DC-SIGN protein present on the surface of the cell, so as to thereby inhibit HCV infection of the cell susceptible to HCV infection. This infection provides a method of inhibiting HCV infection of a cell susceptible to HCV infection which comprises contacting the cell with an amount of a compound effective to inhibit binding of an HCV envelope glycoprotein to a DC-SIGNR protein present on the surface of the cell, so as to thereby inhibit HCV infection of the cell susceptible to HCV infection. Compds. of the present invention inhibit HCV infection of cells susceptible to HCV infection. The compds. of the present invention preferably have specificity for preventing or inhibiting infection by HCV and do not inhibit infection by other viruses, such as HIV, that may utilize DC-SIGN or DC-SIGNR for infection. Moreover the compds. of the present invention preferably do not interfere or inhibit members of the Ig superfamily, in particular, the compds. do not interfere with ICAM-2 or ICAM-3 or with ICAM-2-like, or ICAM-3-like mols.

AN 2003:5672 HCAPLUS <<LOGINID::20080722>>

DN 138:83339

TI Sequences of human C-type lectin DC-SIGN and DC-SIGNR, and uses thereof for inhibiting hepatitis C virus infection

IN Olson, William C.; Maddon, Paul J.

PA Progenics Pharmaceuticals, Inc., USA

SO PCT Int. Appl., 166 pp. CODEN: PIXXD2

DT Patent

LA English

LA English

FAN.	CNT	1																	
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PI		2003									WO 2	002-		20020626					
	WO	2003	0000:	24		A3		2003	0731										
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	US	2003									US 2001-891894						0010	626	
	CA	2452	049						CA 2002-2452049						20020626				
	AU	2002	3244	61					AU 2002-324461										
	EP	1411	980			A2		2004	0428	EP 2002-759107						2	0020	626	
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L29 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2008 ACS on STN

TI Aromatic alpha-glycosides of mannose are powerful inhibitors of the adherence of type I fimbriated Escherichia coli to yeast and intestinal epithelial cells

- AB Adherence of bacteria via their surface lectins to host epithelial cells is considered an important initial event in bacterial pathogenesis. Mannose-specific (type 1) fimbriae are among the most commonly found lectins in enterobacteria. The effect of aromatic α -glycosides of mannose was studied on the agglutination of mannan-containing yeasts by different strains of E. coli and on the adherence of the bacteria to quinea pig ileal epithelial cells. In both systems, these compds. were considerably more effective inhibitors than Me α -mannoside, with 4-methylumbellifervl \alpha-mannoside and p-nitro-o-chlorophenyl a-mannoside being the strongest inhibitors. Both compds. were approx. 400-times stronger inhibitors of yeast agglutination by E. coli 0128 than was Me α-mannoside and 1000- and 470-fold stronger, resp., than was Me α -mannoside in inhibiting the adherence of the bacteria to ileal epithelial cells. 4-Methylumbelliferyl a-mannoside was 540-1000 times more effective in inhibiting yeast agglutination by 4 addnl. strains of mannose-specific E. coli. It was also more efficient than Me a-mannoside in removing adherent E. coli 0128 from ileal epithelial cells. The results provide further evidence that type 1 fimbriae of E. coli possess a hydrophobic region next to the mannose-binding site. The results suggest that 4-methyllumbelliferyl α-mannoside and p-nitro-o-chlorophenyl α-mannoside are good candidates for the design of therapeutic agents that may prevent adherence in vivo and infection by E. coli strains that express type 1 fimbriae.
- AN 1987:135142 HCAPLUS <<LOGINID::20080722>>
- DN 106:135142
- OREF 106:21986h,21987a
- TI Aromatic alpha-glycosides of mannose are powerful inhibitors of the adherence of type 1 fimbriated Escherichia coli to yeast and intestinal epithelial cells
- AU Firon, Nurit; Ashkenazi, Shai; Mirelman, David; Ofek, Itzhak; Sharon, Nathan
- CS Dep. Biophys., Weizmann Inst. Sci., Rehovot, Israel
- SO Infection and Immunity (1987), 55(2), 472-6
- CODEN: INFIBR: ISSN: 0019-9567
- DT Journal
- LA English
- L29 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2008 ACS on STN
- TI Transport of sugars and amino acids in the intestine: evidence
- for a common carrier
- Rings of everted hamster small intestine were incubated with cycloleucine-14C (I) or L-tyrosine. D-Galactose (II) and L-arginine (III) were partially competitive inhibitors of I transport; neutral amino acids were fully competitive inhibitors. Kinetic consts. for I transport were calculated Straight line and parabolic curves were seen with neutral amino acids and II and III, resp. I, histidine, proline, and methionine are thought to share a common binding site in the membrane. If II and III are allosteric inhibitors of the transport of neutral amino acids, probably 3 different substrate-binding sites (1 each for sugars, neutral amino acids, and basic amino acids) plus the Na+-binding site, are closely associated in the membrane, as in a mosaic. The actively transported analogs of II and III inhibited the transport of neutral amino acids. Of the basic amino acids, L-lysine was as effective as III, and L-ornithine more so. Of the sugars, II was the strongest inhibitor, but other metabolizable and nonmetabolizable actively transported sugars were inhibitory. Compds. not actively transported were inert such as methyl-a-glucoside, methyl-a -mannoside, D-fructose,

L-sorbose, and 2-deoxy-D-galactose. Phlorizin was a poor inhibitor of amino acid transport, and its presence prevented the inhibitory effects of II. Nonmetabolizable sugars had qual. identical effects as II. D-hlose, with an axial --OH group as in II, was more inhibitory than glucose. For

- I and tyrosine, methyl- α -D-mannopyranoside > D-glucose > 6-deoxy-D-glucose > 3-O-methyl-D-glucose > methyl- α -D-glucopyranoside > D-glucopyranoside > D-glucopyranoside > D-glusopyranoside > D-glusopyranoside > D-glusopyranoside > D-glusopyranoside > D-glusopyranoside > D-glucopyranoside > D-glucopyran
- AN 1966:78165 HCAPLUS <<LOGINID::20080722>>
- DN 64:78165
- OREF 64:14693d-q
- TI Transport of sugars and amino acids in the intestine: evidence
- for a common carrier AU Alvarado, Francisco
- CS Chicago Med. School
- SO Science (Washington, DC, United States) (1966), 151(3713), 1010-13 CODEN: SCIEAS; ISSN: 0036-8075
- DT Journal
- LA English